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INTERNAL FIXATION OF FRACTURES OF THE NECK OF THE FEMUR

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A fracture of the neck of the femur is a serious injury and to the aged person it is practically a death blow. Because of the teachings of two midwestern surgeons, Ruth and Maxwell, and as a result of the work of Royal Whitman, the medical profession a generation ago became aware that union could be obtained after this type of fracture in a fair proportion of cases if rational and efficient treatment was promptly instituted. Sufficient data have now been gathered to enable one to evaluate Royal Whitman's method,[1] which is almost universally used in America. A commission appointed by the American Orthopaedic Association [2] reported that bony union was secured by the Whitman method in about 50 per cent of the cases in which the patients were less than 60 years of age, but that in cases in which the patients were more than 60 there was a sharp decrease in the percentage of good results. The mortality incident to the accident and treatment varied, but the average was about 15 per cent. Furthermore, the report definitely indicated that results were better where some form of internal fixation was provided.

The reasons for such unsatisfactory results following conservative measures are many. It is not sufficient to blame the advanced age of the patient, because other fractures in an elderly person unite readily.

ANATOMIC STRUCTURE

The crux of the situation rests in the anatomic structure of the neck and head of the femur. The proximal fragment cannot be controlled because there is no way of grasping it, so the lower fragment must be brought into alinement and maintained there. If the fragments

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1. Whitman, Royal: The Abduction Method, Considered as the Exponent of a Treatment for All Forms of Fracture at the Hip in Accord with Surgical Principles, Am. J. Surg. **21**:335-344 (Sept.) 1933.

2. Report of a Commission Appointed by the American Orthopaedic Association to Study the End Results of Intracapsular Fractures of the Neck of the Femur, J. Bone & Joint Surg. **12**:966-969 (Oct.) 1930.

are not securely held in this position, the shearing force that is produced by the pull of the powerful pelvic and femoral muscles and by the angle that the neck of the femur forms with the shaft, which is about 127 degrees, will either cause angulation at the site of fracture or cause the lower fragment to slip upward. The capsule of the hip joint may be so torn that it will lie between the fragments. This is probably a rare happening, but I have found it when the joint was opened in several cases. Gaenslen [3] recently reported a series of experiments in which fractures of the hips of dogs were produced by arthrotomy. Although the capsule was tucked in and held by sutures between the fragments, bony union was secured if the fragments were spiked together with knitting needles. His only failure to obtain union was in an animal in which he used Kirschner's wires instead of the more rigid knitting needles to provide fixation. The wires failed to maintain fixation, and Gaenslen [4] concluded that the failure to maintain fixation led to nonunion.

BLOOD SUPPLY

The blood supply may be interfered with by the trauma incident to the accident. The blood supply of the head and neck of the femur is derived from four sources: the diaphysis, the epiphysis, the ligamentum teres femoris and the periosteum. The vessels which come from the diaphysis cross the epiphysial line to enter the head of the femur. The epiphysial blood vessels enter the head of the femur, along the edge of the articular surface, close to the epiphysial junction. Two groups of epiphysial vessels are recognized: One group comes from the subtrochanteric fossa and enters the head of the femur from above; the other group comes from the region about the insertion of the articular capsule on the inferior aspect of the neck of the femur and enters the lower end of the head of the bone. Blood vessels are carried by the ligamentum teres femoris. It is probable that the blood supply derived from the ligamentum teres femoris is so meager as to be of no value in about 25 per cent of elderly persons.[5] In the new-born a free blood supply through this ligament is always present. Kolodny [6] expressed the belief that the periosteal group of vessels were important, and he satisfied himself that the portion of the neck of the femur lying within

3. Gaenslen, F. J.: Fracture of the Neck of the Femur, J. A. M. A. **107**: 105-114 (July 11) 1936.

4. Gaenslen, F. J.: Subcutaneous Spike Fixation of Fresh Fractures of the Neck of the Femur, J. Bone & Joint Surg. **17**:739-748 (July) 1935.

5. Wolcott, W. E.: Circulation of the Head and Neck of the Femur: Its Relation to Nonunion in Fractures of the Femoral Neck, J. A. M. A. **100**:27-33 (Jan. 7) 1933.

6. Kolodny, Anatole: The Architecture and the Blood Supply of the Head and Neck of the Femur and Their Importance in the Pathology of Fractures of the Neck, J. Bone & Joint Surg. **7**:575-597 (July) 1925.

the capsule possessed a periosteum which differed little, if at all, from the periosteum of the extracapsular portion of the neck of the bone. Kolodny stated that the diaphysial, periosteal and epiphysial blood vessels were the important ones in the nutrition of the head and neck of the femur of an elderly person. Therefore, it would seem that sufficient blood is supplied to the fractured bone to ensure bony repair if proper apposition of the fragments is maintained.

REDUCTION AND FIXATION

The surgeon, and particularly the inexperienced one, may have difficulty not only in reducing a fracture of the neck of the femur but in providing fixation after reduction. Manipulative reduction should always be checked by roentgenograms, which should be made in the lateral as well as in the anteroposterior position, if possible. Proper reduction and fixation are the essential factors, and failure to provide the latter has led to many poor results.

ASEPTIC NECROSIS

Aseptic necrosis frequently is blamed for the failure of the fracture to unite. Gaenslen, in advocating internal fixation in the treatment of this type of fracture, said that the cause of aseptic necrosis, which too commonly is encountered in unimpacted fractures, is mobility at the site of the fracture. Absolute immobility in any cast is impossible of attainment, and if there is any great amount of absorption at the line of fracture one can readily understand how displacement of the fragments would occur.

SELECTION OF PATIENTS

Unfortunately, every patient who has a fracture of the neck of the femur cannot endure the necessary treatment. The debilitated elderly person who perhaps is suffering from the terminal stage of a constitutional disease may not be a fit subject for the ordeal, and the extremely old and feeble person who is not suffering from any well defined constitutional disability is maintaining himself on such a narrow margin that, as stated before, the accident is nearly always his death blow. The prolonged fixation in a cast incident to the Whitman method has militated more than any other factor against the carrying out of rational treatment. If the wearing of a cast for such a long period could be avoided, or if the time could be materially shortened, any patient who has enough reserve to tolerate a major surgical procedure would be able to withstand the treatment of a fracture of the neck of the femur.

OPERATIVE TREATMENT

In the hope of shortening the period of confinement and inactivity, operative fixation has been advanced at various times, only to be abandoned because no systematic study was made of the end-results in cases

in which the method was used. The poor results of a method in its developmental stage are always viewed with critical eyes and may overwhelm a sound rational procedure, which, if persistently advanced and improved, would hold its place in the surgical armamentarium. Bad news travels as fast in the surgical world as it does in other walks of life.

The Smith-Petersen Nail.—Smith-Petersen,[7] of Boston, in 1931 reported the use of a triple flanged metal nail in the treatment of fractures of the neck of the femur. The results with this method were so good that attention was promptly attracted to it. Johansson,[8] of Sweden, and King,[9] of Australia, have advocated the use of the Smith-Petersen flanged nail, but they use a cannulated nail so that it can be driven in over a guiding wire. The literature on the subject is increasing. Thomas,[10] Knowles,[11] Gaenslen,[4] Moore,[12] Wescott,[13] W. Meyer [14] and others, including myself, have reported fractures of the neck of the femur which have been treated by internal fixation and have described the technic of the method. Sufficient data have now been recorded to prove that the results with this method are quite good.

Impacted Fractures.—Too often, the surgeon has been lulled into a false sense of security in the treatment of a fracture of the neck of the femur, because the roentgenogram has dissolved what is termed "impaction" of the fragments. The impaction too frequently breaks down and, except in a rare instance, should not be trusted. The rare instance I refer to is the case in which the fracture is situated close to, or involves, the head of the femur; there is really a compression

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7. Smith-Petersen, M. N.; Cave, E. F., and Vangorder, G. W.: Intracapsular Fractures of the Neck of the Femur: Treatment of Internal Fixation, Arch. Surg. **23**:715-759 (Nov.) 1931.

8. Johansson, Sven: On Operative Treatment of Medial Fractures of the Neck of the Femur, Acta orthop. Scandinav. **3**:362-392, 1932.

9. King, Thomas: Recent Intracapsular Fractures of the Neck of the Femur: A Critical Consideration of Their Treatment and a Description of a New Technique, M. J. Australia **1**:5-15 (Jan. 6) 1934.

10. Thomas, T. T.: Fixation by a Wood Screw Without Arthrotomy in Certain Fractures of the Neck of the Femur, Am. J. Surg. **35**:292-295 (Sept.) 1921.

11. Knowles, F. L.: Fractures of the Neck of the Femur, Wisconsin M. J. **35**:106-108 (Feb.) 1936.

12. Moore, A. T.: Fracture of the Hip Joint (Intracapsular): A New Method of Skeletal Fixation, J. South Carolina M. A. **30**:199-205 (Oct.) 1934.

13. Wescott, H. H.: Preliminary Report of a Method of Internal Fixation of Transcervical Fractures of the Neck of the Femur in the Aged, Virginia M. Monthly **59**:197-204 (July) 1932; A Method for the Internal Fixation of Transcervical Fractures of the Femur, J. Bone & Joint Surg. **16**:372-378 (April) 1934.

14. Meyer, W.: Result of Nailing the Head to the Neck in Intracapsular Fracture of the Left Femur, with Excellent Functional Result, Tr. New York Acad. Med. **10**:672, 1893.

fracture of the head and the upper portion of the neck of the femur (fig. 1*a*). An impacted fracture through the middle of the neck of the femur (fig. 1*b*) or near the greater trochanter should practically always be broken up with the patient anesthetized and the fracture reduced by manipulation. Adequate fixation must then be provided in a plaster of paris cast or by aid of some form of internal fixation.

Methods of Internal Fixation.—Smith-Petersen advised open reduction of the fracture and insertion of the nail by visual supervision. Reduction of the fracture by arthrotomy is truly a major operative procedure and one that should be undertaken only by experienced surgeons. With the so-called "blind" method, the fracture is reduced by

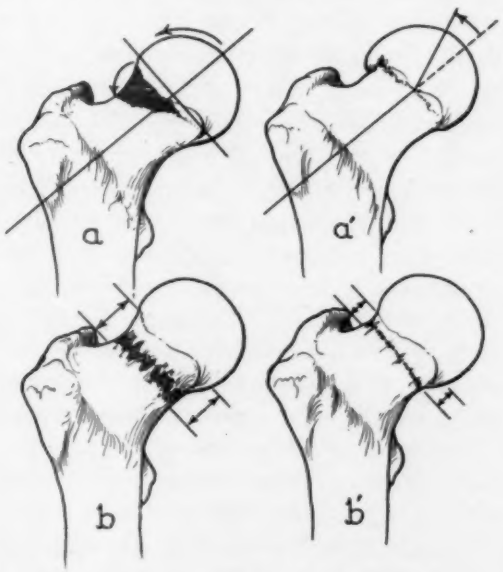

Fig. 1.—In *a* is shown a fracture of the neck of the femur, the darkened area indicating the region of compression; *a'* the position after compression; in *b*, a typical transverse fracture of the neck of the femur, the darkened area showing the region of absorption, and in *b'* union with shortening of the neck of the bone.

one of the recognized manipulative procedures, and after roentgenographic or fluoroscopic examination reveals that reduction has been secured a small incision is made over the greater trochanter and the nail driven through the neck and well into the head of the femur. The "blind" method can be made more accurate by inserting a guiding wire after the apposition of the fragments is known to be accurate and by checking the position of the wire by anteroposterior and lateral roentgenograms. If the position of the wire is satisfactory as to direction and depth, a cannulated flanged nail is passed over it and driven through the neck and into the head of the femur. The wire is then withdrawn.

Insertion of the nail in this manner is not as certain to be accurate as one might expect, because even when the nail is guided by the wire it may wander and bend the wire, particularly as it enters the head of the femur. If the wire is bent to any degree, the end may break off when the wire is withdrawn through the cannulated nail. To prevent this the wire should be passed completely through the head of the femur and should extend into the acetabulum. This steadies the head of the femur as the nail crosses the line of fracture and is driven into the head of the bone. The insertion of the wire and the roentgenographic control of the position is often a prolonged procedure, for the roentgenograms have to be developed. The operation is remarkably safe if done with the patient under spinal anesthesia, for there is practically no dissection or exposure of tissue and so, no shock. I have had no experience with the reduction of such fractures under fluoroscopic control.

Objectionable Features of the Nail.—The flanged nail of Smith-Petersen is strong and in theory, at least, would seem to solve the problem of the treatment of this type of fracture. It at first was hoped it would not be necessary to apply a cast and that weight bearing could be permitted very early, but weight bearing, if permitted before bony union is well under way, has proved disastrous. It is advisable for the patient to wear a cast for from two to six weeks and permit no weight bearing for at least three months. The triple flanged nail of Smith-Petersen has a large area of metal which comes in contact with the bone, and in some instances it is poorly tolerated. Although the nail may have been driven in securely and may have been in excellent position, it may become loose as a result of absorption of bone about it and back out, as it were. How to prevent this is a problem as yet unsolved. Absorption at the line of fracture also occurs in some cases, and as the neck of the femur becomes shorter, the nail, which is firmly embedded in the head of the bone, protrudes beyond the trochanter and becomes loose.

Fixation with a Lag Screw.—Because of this difficulty, at the Mayo Clinic the use of what is called a "lag" screw has been resorted to. This will hold the fragments together. I claim nothing original for the use of the lag screw, as carpenters have long used it in cabinet work when they desired to coapt pieces of wood and it was undesirable to insert a bolt. I do not believe that it has been used previously in just this manner for the fixation of fractures of the hip, though M. O. Henry described a screw bolt similar in principle. It has been used in four cases at the clinic; in one of these cases the fracture was old, and in the others the fractures were recent. Successful results were obtained in three cases; in one case the time which has elapsed since the operation has not been sufficient to permit one to determine the end-result. The

threads in the portion of the screw that goes into the head of the femur are of the wood-screw type; large and little notches are placed along the thread so that it will hold better (fig. 2). The lag screw which has been used at the clinic is cannulated and is inserted over a guiding wire.

DATA IN SIXTEEN CASES IN WHICH INTERNAL FIXATION WAS EMPLOYED

Sex and Age.—Of a group of sixteen patients with fracture of the neck of the femur who were treated with internal fixation, four were men and twelve were women. The distribution of these patients according to the various age groups follows: between 20 and 29 years,

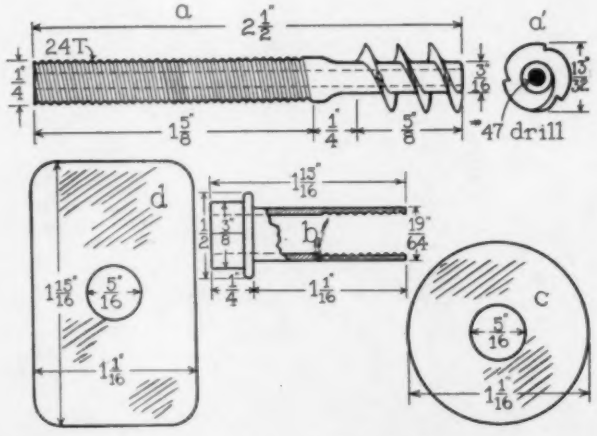

Fig. 2.—Diagram of a lag screw: *a*, the lag screw; *b*, the sleeve nut; *d* and c washers, and *a'*, the end view, showing cannulate opening.

one; between 40 and 49 years, one; between 50 and 59 years, four; between 60 and 69 years, five; between 70 and 79 years, four, and between 80 and 89 years, one. Thus, ten of the patients were more than 60 years of age.

Selection of Patients for Operation.—Any patient who was thought fit to withstand a major surgical operation was subjected to the procedure. The majority of patients were operated on as soon as the general examination was completed, and all were subjected to operation within three weeks of the accident. King, of Melbourne, Australia, prefers to make the patient comfortable and to wait three weeks before operating. By the end of that time he has a good idea as to the fitness of the patient for operation.

Anesthesia.—Spinal anesthesia is preferable; this type of anesthesia was used in eleven of the sixteen cases.

Type of Operation.—Arthrotomy was done in three cases. This permits visual supervision of the reduction of the fracture and the insertion of the nail. In the remaining thirteen cases closed reduction was done by Leadbetter's [15] method, and the nail or screw was inserted along a Kirschner wire, which was used as a guide. A cannulated Smith-Petersen nail was used in thirteen cases, a cannulated plain screw with a large thread in one case and the cannulated lag screw in two cases. The roentgenographic examination at the time of reduction showed perfect and complete reduction in nine cases. In seven cases there was overcorrection of the deformity; that is, the head of the femur was in a valgus position, which really is a desirable position, for I cannot recall

Fig. 3.—Apparatus for moving the hip.

that I ever have seen such a fracture overcorrected in this manner fail to unite. Preoperative classification of the fractures disclosed that one was oblique, five were transverse, and nine were impacted. All the fractures were in the neck of the femur; no trochanteric fractures are included in this series.

External Fixation and Institution of Motion.—In treatment of these patients at the clinic either a single spica cast, which extended downward to the knee or a double spica, which extended from the thorax to the toes on the affected side and to the knee on the sound side, was used. The cast was bivalved or was removed in two weeks so as to permit bending of the knee, unless there was some complicating factor. Motion was instituted by aid of a sling placed under the knee, and this was

15. Leadbetter, G. W.: A Treatment for Fracture of the Neck of the Femur, J. Bone & Joint Surg. **15**:931-940 (Oct.) 1933.

pulled by the patient by means of a rope which passed through a pulley on an overhead bar (fig. 3). By this contrivance the hip is moved as the patient moves the knee.

Weight Bearing.—The patients, on the average, were allowed up on crutches in seven weeks, and partial weight bearing was permitted. Full weight bearing without support of any kind was permitted, on the average, in four and a half months (fig. 4*A*).

Days Spent in Hospital.—The number of days spent in the hospital averaged sixty-eight.

Removal of the Nail.—When union is complete the nail had better be removed (fig. 4*B*). However, one patient who was past 80 years

Fig. 4.—*A*, fracture of the hip united three and a half months after insertion of a lag screw. *B*, union of the fracture of the hip shown in *A*. The roentgenogram was made after removal of the lag screw.

of age lived comfortably for two years with good function in spite of the fact that the nail was never removed.

RESULTS

There was no postoperative death or serious infection, although low grade infection developed late in two cases. Solid bony union has been secured in thirteen of the sixteen cases; one fracture definitely has failed to unite, and in two cases the results are as yet indeterminate. Therefore, of fourteen cases in which sufficient time has elapsed to be

certain of the result, in thirteen, or 92.8 per cent, bony union has been obtained. In the six cases in which the patient was less than 60, the fractures united solidly. In two of the ten cases in which the patient was more than 60, the results are still indeterminate; that is, bony union is not demonstrable in the roentgenogram. I feel confident that one of these fractures will unite, but I am not so certain about the other one. In one case the result may be classified definitely as a failure. Bony union therefore occurred in seven, or 87.5 per cent, of the eight cases in which the convalescence had progressed far enough to enable one to determine the end-result.

Results of Internal Fixation in Sixteen Cases of Fracture of Neck of Femur

1. Entire group of cases	16
Deaths following operation	0
Low grade infections (late)	2
Patients still under observation	2
Cases in which nonunion occurred	1
Cases in which bony union occurred	13*
2. Patients less than 60	6
Cases in which bony union occurred	6
3. Patients more than 60	10
Cases in which nonunion occurred	1
Patients still under observation	2
Cases in which bony union occurred	7†

* This number represents 92.8 per cent of the fourteen cases in which end-results could be determined.

† This number represents 87.5 per cent of the eight cases (group 3) in which the end-results could be determined.

COMMENT

The reason for the decided interest shown in the internal fixation of fractures of the neck of the femur is that the results following the conservative treatment were on the whole unsatisfactory.

The results obtained in this limited number of cases at the clinic are far better than were ever obtained there in a similar group of cases in which the fractures were treated conservatively. The sharp pains that occur commonly after conservative reduction are absent in cases in which the fracture is treated by internal fixation, because the fixation is absolute. Many of the patients do not have the slightest pain. Furthermore, when internal fixation is employed the patient is much more comfortable during convalescence. The discomfort that the patient endures when he tries to limber up after he has spent three months in a cast is not encountered because early mobility of knee and hip is possible when internal fixation is employed. The effect on the morale of the patient is an exceedingly happy one. There were no deaths in the

series of cases in which this type of treatment was employed at the clinic. I believe the danger of pulmonary embolism is decreased because the patients can be moved about early, active motion is permitted and the blood is kept circulating in the iliac vessels.

Pitted against these favorable aspects are an array of objections which must be considered. The insertion of the Smith-Petersen nail, the lag screw, pins or a bone graft is not easy, and the surgeon too often is chagrined and startled to find how far wrong his aim was in the insertion of these objects. This has been acknowledged by all and has led to the manufacture of many gadgets and instruments designed to overcome this difficulty. The specter of infection always hovers about these procedures. An infected hip means either death or prolonged drainage with all its discomforts, and experience with such a condition is sufficient to dampen the ardor of even the most enthusiastic surgeon. This type of operation must be done only in a properly equipped hospital and by an experienced surgeon. The nail does not always stay where it is put, for absorption about the nail may cause it to become loose and slide outward. Absorption of the neck of the femur at the site of fracture may so shorten the neck of the bone that even if the end of the nail stays in the head of the femur the head of the nail may protrude considerably beyond the trochanter and the action of the muscles during movement may further tend to loosen it. It takes at least three months for bony union to occur, and even with perfect internal fixation, full weight bearing cannot be permitted until roentgenographic examination discloses that bony union is present. While it is true that a larger percentage of patients with fractures of the neck of the femur can be treated by this method than by the Whitman method, because prolonged wearing of a cast is not necessary, if internal fixation is used indiscriminately in all cases of fracture of the neck of the femur, failures and disasters will follow and will tend to discredit the method. The incidence of fractures of the hip compared to other fractures is relatively small, so that the experience of any one man is meager unless he is in a surgical center and by agreement the patients are referred to him; therefore, the operative treatment of these fractures should be centralized and experience provided. Time will disclose whether flattening and distortion of the head of the femur are less frequent following the use of internal fixation than they are following the use of the closed method.

SUMMARY

I believe that internal fixation of fractures of the neck of the femur gives better results with no higher mortality than does conservative

treatment. Therefore, internal fixation should be employed when it can be carried out in the proper surroundings and by a surgeon of sufficient training in surgical operations on the bones. Lacking such conditions, it is better either to refer the patient to a hospital where the exacting conditions can be fulfilled or to treat the patient by such conservative methods as the surgeon is familiar with. There is no hurry in carrying out internal fixation. One authority, King, of Australia, prefers to wait three weeks and thus escape the high early mortality that is encountered in conjunction with this serious injury.

CARCINOMA OF THE GALLBLADDER

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Carcinoma of the gallbladder, a rare condition in 1850, has since become a common one. The apparent increase in the incidence of the disease roughly parallels the increase in the frequency of operations on the gallbladder. The purpose of this paper is to add to the literature the data on 48 cases of carcinoma of the gallbladder observed at the New York Hospital from 1915 to 1935. After a brief historical review, the cases are analyzed pathologically, the pathogenesis being emphasized whenever possible. The same cases are then considered clinically in an attempt to clarify the clinical picture and thereby to aid in early diagnosis.

HISTORICAL REVIEW

In 1777 Maximilian Stoll,[1] of Vienna, published the first authentic record of carcinoma of the gallbladder, reporting 2 cases in which autopsy was performed. His first case not only introduced a new group of diseases but was unique, the patient having situs inversus viscerum, with carcinoma of the left-sided gallbladder. Hallé [2] in 1786 reported the next case, followed by Baillie's [3] case in 1794, the authenticity of which has been questioned.

From 1800 to 1850 but 9 cases were reported, in 2 of which the lesions probably were secondary. The first full account appeared in this period—the case of Durand-Fardel [4] in 1838.

From 1850 to 1860 9 more cases were recorded. Heschl [5] in 1852 reported 1 case and referred to 2 others which he had observed, both of which were associated with cholelithiasis.

From the Department of Surgery of the New York Hospital and Cornell Medical College.

The Department of Pathology of the New York Hospital furnished the autopsy material herein presented.

1. Stoll, Maximilian: Rations medendi, in Nosocomio practico Vindobonensi, Vienna, A. Bernardi, 1777, pt. 1, p. 290.

2. Hallé, Jean Noël: Histoire de la société de médicin, 1786 (Paris, 1790), p. 125.

3. Baillie, M.: Anatomie des krankhaften Baues von einigen der wichtigsten Theile im menschlichen Körper. Aus dem Englischen mit zusätzen von S. T. Sömmering, Berlin, Leopold Voss, 1794.

4. Durand-Fardel, C. L.: Note pour servir à l'histoire des maladies de la vésicule biliare, Bull. Soc. anat. de Paris **13**:157-160, 1838.

5. Heschl, R.: Ein Fall von Zottenkrebs der Gallenblase, Ztschr. d. k.-k. Gesellsch. d. Aerzte zu Wien **2**:251, 1852.

The decade from 1860 to 1870 enriched the literature by 15 cases. In 1870 Villard [6] collected reports of 26 cases, contributing the most complete study up to that time. Musser [7] in 1889 analyzed 100 cases; Courvoisier [8] in 1890 devoted a chapter in his "Pathologie und Chirurgie der Gallenwege" to a summary of 103 cases; Ames [9] in 1894 published his treatise, and carcinoma of the gallbladder, though uncommon, ceased to be rare.

Whereas nineteenth century writers acquainted one with the clinical and the pathologic picture of the disease, it remained for authors of the present century to emphasize its frequency. Notable contributors to the subject since 1900 are Sherrill,[10] Robson,[11] Moynihan,[12] Cotte,[13] Mayo,[14] Quenu,[15] Rolleston,[16] Smithies,[17] Fawcett and Rippmann,[18] Deaver,[19] Lentze [20] and recently Illingworth.[21] It is estimated that at least 2,000 cases have now been reported in the literature.

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6. Villard, Ferdinand: Etude sur le cancer primitif des voies biliares, Bull. Soc. anat. de Paris **44**:217, 1869; Paris, A. Delahaye, 1870.

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8. Courvoisier, L. G.: Casuistisch-statistische Beiträge zur Pathologie und Chirurgie der Gallenwege, Leipzig, F. C. W. Vogel, 1890.

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11. Robson, A. W. M.: Adenoma of the Gall-Bladder, Med.-Chir. Tr., London **88**:229-231, 1905.

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13. Cotte, G.: Traitement chirurgicale du cancer des voies biliares, Rev. de chir. **39**:1135-1152, 1909.

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16. Rolleston, H. D., and McNee, J. W.: Diseases of the Liver, Gall-Bladder, and Bile-Ducts, ed. 3, New York, The Macmillan Company, 1929.

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18. Fawcett, J., and Rippmann, C. H.: Carcinoma of the Gall-Bladder Associated with Gall-Stones, Guy's Hosp. Rep. **67**:41-80, 1913.

19. Deaver, J. B.: Carcinoma of the Gall-Bladder, M. Rec. **96**:47-49, 1919.

20. Lentze, F. A.: Gallensteine und Gallenblasenkarzinom, Beitr. z. klin. Chir. **137**:38-62, 1926.

21. Illingworth, C. F. W.: Carcinoma of the Gallbladder, Brit. J. Surg. **23**: 4-18, 1935.

INCIDENCE

This glimpse of the history of the disease fails to convey a concept of the frequency of carcinoma of the gallbladder. It probably is impossible, in the absence of compulsory necropsies, to relate its true incidence. The most reliable sources of information are large series of autopsies.

To estimate the frequency of the disease on the basis of cases in which operation was performed gives a distorted view, for these are selected. This fact may contribute to the general difference in attitude between the surgeon and the internist toward disease of the gallbladder. It certainly represents the origin of widely differing opinion concerning the frequency of carcinoma of the gallbladder. Table 1 shows the incidence of the disease in several large series of operations and autopsies. The surgical incidence, it will be noted, is more variable

TABLE 1.—*Incidence of Carcinoma of the Gallbladder at Operation and Autopsy*

Author	Operations on Gallbladder	Autopsies	Carcinoma of Gallbladder	Incidence, Percentage
Erdmann: New York M. J. **111**: 705-707, 1920	224	.....	15	6.70
Illingworth [21]	1,057	.....	34	3.20
Smithies [17]	1,000	.....	23	2.30
Deaver [19]	1,000	.....	16	1.60
Judd and Gray: Surg. Gynec. & Obst. **55**: 308-315, 1932	22,365	.....	212	1.00
Illingworth [21]	.....	8,490	36	0.42
Fawcett and Rippmann [18]	.....	592	48	0.81
von Berencsy and von Wolff: Ztschr. f. Krebsforsch. **21**: 109-118, 1924	.....	19,908	300	1.50

and several times greater than the autopsy incidence in the experience of many authors.

The data in the 48 cases of the New York Hospital were compiled from surgical and autopsy material. Forty-five cases were found in a group of 1,500 operations on the gallbladder, a surgical incidence of 3 per cent. The remaining cases were not discovered at operation. Of the 2,941 autopsies performed between 1915 and 1935, 18 showed carcinoma of the gallbladder, an autopsy incidence of 0.61 per cent.

AGE AND SEX DISTRIBUTION

The age incidence of carcinoma of the gallbladder closely corresponds to that of cancer as a whole. Most cases occur in persons between the ages of 50 and 60. It is interesting to note that inflammatory disease of the gallbladder is most common in persons between 40 and 50, some ten years earlier.

The youngest patient on record is a 22 year old boy (Proescher [22]). Several cases are recorded in persons in the third decade. The oldest patient was mentioned by Illingworth [21] as being 95.

22. Proescher, F.: A Remarkable Case of Carcinoma of the Gallbladder in a Man Twenty-Two Years Old, J. A. M. A. **48**:481-483 (Feb. 9) 1907.

The age distribution in several groups of cases is shown in table 2. The average age in this series is 53.6 years; the youngest patient was 28 and the oldest 85.

Carcinoma of the gallbladder is more common in women than in men, the ratio being roughly 4:1. The proportion probably is due to the preponderance of cholelithiasis in women. In most series from 80 to 90 per cent of the patients are women. In this group 77 per cent were women.

ETIOLOGY

Probably there is no truer concept of the etiology of carcinoma of the gallbladder than of cancer of any other organ. Cohnheim's theory of embryonic rests has been invoked, especially to explain squamous carcinoma of the gallbladder. Some English authors have thought heredity significant. Multiple papillomas of the gallbladder have been

TABLE 2.—*Age Distribution in Several Groups of Cases*

Years	All Cancer,* Percentage	Judd and Gray's,† Percentage	Author's Series Percentage
1–9	0.5	....	...
10–19	0.5	....	....
20–29	1.3	0.9	2.0
30–39	4.6	4.5	4.2
40–49	12.6	14.9	25.4
50–59	21.7	40.3	31.2
60–69	27.1	33.1	20.8
70–79	23.1	6.0	4.2
80–89	7.5	....	2.0
90–99	0.6	....	....

* United States Mortality Statistics, 1932, pp. 134-135.
† Surg., Gynec. & Obst. 55: 308-315, 1932.

considered precancerous. The mass of evidence on the incidence of cholelithiasis in cancer of the gallbladder established chronic irritation as a more likely predicant. Experimental studies, while inconclusive, support this theory.

Relation of Cholelithiasis.—In Musser's cases of carcinoma of the gallbladder the incidence of cholelithiasis was 69 per cent; Janowski's [23] series represented the other extreme, with an incidence of 100 per cent. In the 890 cases reviewed by Lentze [20] in 1926, the incidence of cholelithiasis was 83 per cent. In the remaining 17 per cent, carcinoma was superimposed on inflammatory lesions. In the present series the incidence of cholelithiasis was 79 per cent. Lancereaux [24] and others expressed the belief that stones are formed secondary to carcinoma. The weight of evidence is contrary to this view. In 48 per cent of the cases at the New York Hospital there was a history of colic of long duration,

23. Janowski, W.: Ueber Veränderungen in der Gallenblase beim Vorhanden sein von Gallensteinen, Beitr. z. path. Anat. u. z. allg. Path. **10**:449-480, 1891.

24. Lancereaux, M.: Du cancer de la vésicule biliare, Semaine méd. **7**:334, 1887.

and many of the stones were of the calcified type, which are known to form slowly. Furthermore, Graham [25] collected reports of 38 cases of metastatic carcinoma of the gallbladder in which the incidence of cholelithiasis was only 8 per cent, a figure which approaches the incidence of simple cholelithiasis for this age group. Such observations are prima facie evidence that cholelithiasis predisposes to carcinoma of the gallbladder.

Kazama [26] in 1922 was the first to test this relation experimentally. He inserted foreign bodies (stones, sutures and mucosa) into the gallbladders of guinea-pigs and found that after a time carcinoma of the gallbladder, even a metastasizing tumor, developed in some of the animals. His researches stimulated a series of similar experiments by Leitsch,[27] Clemente [28] and others, the results of which are controversial. Lazarus-Barlow,[29] who demonstrated radio-activity in the salts of some gallstones, suggested that this radiation caused the malignant growth.

Burrows [30] recently reviewed the subject of the experimental production of carcinoma of the gallbladder. After cautious study, he concluded that cancer has not been produced and that the numerous attempts have failed to demonstrate any carcinogenic substance in gallstones. Foreign bodies in the gallbladder stimulate proliferative reactions in the mucosa that simulate cancer but do not cross the indefinite boundary between metaplasia and neoplasia.

Relation of Infection.—A pathologic change more constant than cholelithiasis is infection. In cases of carcinoma in the early stage in which only a small portion of the mucosa is invaded by tumor, there are always varying degrees of subacute or chronic infection with fibrous thickening of the free portion of the organ, depicting a long-standing inflammatory process. Consequently, all recent observers have accepted infection as a precursor of carcinoma.

Relation of Papillomas.—In 1910 MacCarty [31] emphasized the frequency of benign papillomas of the gallbladder, describing the con-

25. Graham, E. A.: The Prevention of Carcinoma of the Gallbladder, Ann. Surg. **93**:317-322, 1931.

26. Kazama, Y.: The Studies on the Artificial Production of Tumors in the Viscera, Japan M. World **2**:309-312, 1922.

27. Leitsch, A.: Gallstones and Cancer of the Gall-Bladder, Brit. M. J. **2**: 451-454, 1924.

28. Clemente, G.: Tumori sperimentali della colecisti da catrame, Arch. ital. di chir. **17**:613, 1927.

29. Lazarus-Barlow, W. S.: An Attempt at the Experimental Production of Cancer by Means of Radium, Proc. Roy. Soc. Med. (Sect. Path.) **11**:1, 1917.

30. Burrows, H.: An Experimental Inquiry into the Association Between Gallstones and Primary Cancer of the Gallbladder, Brit. J. Surg. **20**:607-629, 1933.

31. MacCarty, W. C.: Pathology of the Gallbladder and Some Associated Lesions, Ann. Surg. **51**:651-669, 1910.

dition as "cholecystitis catarrhalis papillomatosa." He considered this a type of "strawberry gallbladder," since many of the papillomas contained deposits of cholesterol. This condition has received further recognition from Mayo,[32] Keene,[33] Mölle [34] and Abell.[35]

Malignant degeneration in papillomas has been reported by Ringel,[36] Pels-Leusden [37] and others. Illingworth [21] thought that papillomas rarely show precancerous changes. Phillips,[38] in a series of 500 cases, found but 1 with carcinoma. In this case the papillomas were multiple and all benign, while the carcinoma was solitary and malignant throughout. He could not, then, prove the pathogenesis to be malignant degeneration.

The lesion described by MacCarty occurs relatively early in the course of disease of the gallbladder. All such cases show some infection in the mucosa. Stones were present in 26.8 per cent of Phillips' cases. Because of the deposits of cholesterol, it is likely that many more eventually would have formed stones. The presence of papillomas is ample evidence of the epithelial proliferative power. One cannot, then, deny that the papillomatous gallbladder has the capacity of forming carcinoma, for infection, stones and the power to proliferate are the most common etiologic factors. All that one can require further than this is the ultimate—a susceptible subject.

PATHOLOGIC PICTURE

The pathologic picture of carcinoma of the gallbladder has been described adequately by Courvoisier,[8] Kaufmann,[39] Ewing [40] and others. Pathologically, the disease may be divided into two groups: adenocarcinoma and squamous carcinoma. The adenocarcinomas may be

32. Mayo, C. H.: Papillomas of the Gall-Bladder, in Collected Papers of the Mayo Clinic, Philladelphia, W. B. Saunders Company, 1915, vol. 7, pp. 249-255.

33. Keene, F. E.: Papilloma of the Gall-Bladder, Proc. Path. Soc., Philadelphia **38**:7, 1918.

34. Mölle, H.: Ueber Papillome der Gallenblase, nebst Mitteilung eines selbst beobachteten Falles, Beitr. z. klin. Chir. **99**:173-185, 1916.

35. Abell, I.: Papilloma and Adenoma of the Gallbladder, Ann. Surg. **77**: 276-280, 1923.

36. Ringel: Ueber Papillom der Gallenblase, Arch. f. klin. Chir. **59**:161-166, 1899.

37. Pels-Leusden, F.: Ueber papilläre Wucherungen in der Gallenblase und ihre Beziehungen zur Cholelithiase und zum Carcinom, Arch. f. klin. Chir. **80**:128-160, 1906.

38. Phillips, J. R.: Papilloma of the Gallbladder, Am. J. Surg. **21**:38-42, 1933.

39. Kaufmann, E.: Lehrbuch der speziellen pathologischen Anatomie für Studierende und Aerzte, ed. 5, Berlin, G. Reimer, 1909, vol. 5.

40. Ewing, J.: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1931, pp. 736-740.

subdivided into (*a*) papillary adenocarcinoma, (*b*) infiltrating adenocarcinoma, (*c*) scirrhous adenocarcinoma and (*d*) mucous adenocarcinoma.

Inflammation coupled with the chronic mechanical irritation caused by gallstones produces a variety of reactions in the tissues of the gallbladder. The type of reaction probably determines the type of tumor which is to form. If the submucosa is more responsive to irritation than the mucosa, the resulting papilloma may be composed chiefly of connective tissue elements covered with normal epithelium. In my opinion, this type of papilloma is the least likely to form carcinoma, for the epithelium shows growth restraint under stimulation.

Should the response be an overgrowth of epithelium, simple benign papillomas may develop. With all growth restraint removed and anaplasia in abeyance, the gallbladder may become distended with a bulky, benign papillary growth. Such rare tumors have been described by Sand and Mayer [41] and by Chappet.[42] They are clinically malignant because of their situation but are pathologically benign. Devic and Gallavardin [43] described a case in which the growth extended down into the common duct. In one of the cases in the present series the tumor was definitely of this type, though sufficient connective tissue was present to give it a villous appearance not unlike a bunch of white grapes.

ADENOCARCINOMA

Papillary Adenocarcinoma.—If anaplasia is present, the papillary overgrowth of epithelium is malignant, resulting in papillary adenocarcinoma of the gallbladder. This is a common type; in 12 per cent of the cases at the New York Hospital the condition is of this nature. The tumor is composed of multiple papillary stalks of connective tissue covered with atypical cylindric cells and infiltrated by secondary alveoli lined with cuboidal cells. The tumor may be bulky, sometimes distending the gallbladder or obstructing the cystic duct. Ulceration may limit its growth into the lumen. In general, this type of tumor is slower growing, less malignant and more bulky than any other. Infiltration is slight, metastases are late and infection is marked. Operation is performed in a good many cases for acute or subacute cholecystitis.

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41. Sand, R., and Mayer, L.: Transformation de la vésicule biliare tout entière en un kyste papillafère, Arch. de méd. expér. et d'anat. path. **23**:523-528, 1911.

42. Chappet, V.: Cancer épithélial primitif du canal cholédoque; hydropisie de la vésicule; ictère par rétention; absence de généralisation; cachexie progressive, Lyon méd. **76**:145-157, 1894.

43. Devic and Gallavardin, L.: Etude sur le cancer primitif des canaux biliaires, cholédoque, hépatique et cystique, Rev. de méd. **21**:557, 1901.

Infiltrating Adenocarcinoma.—If anaplasia is marked and ulceration present from the start, papillary overgrowth may never have a chance to occur. The result is infiltrating adenocarcinoma, which formed 56 per cent of this series. The tumor consists of infiltrating atypical alveoli and pseudo-alveoli and does not increase the bulk of the organ. It rapidly invades all layers of the gallbladder and the liver and through adhesions, the duodenum, stomach, colon, omentum and peritoneum. Metastases occur early to the regional nodes and liver. It is in this group that metastases occasionally give rise to the first clinical manifestations of disease.

Scirrhous Adenocarcinoma.—This tumor also is of the infiltrating type of adenocarcinoma, the difference being the extensive growth of new cellular connective tissue. Whether this desmoplastic quality is reaction to preexisting inflammation or to the growth of the tumor is disputed. The picture is that of extensive fibrosis infiltrated by strands of atypical epithelial cells which tend to form alveoli. The fibrosis can be considered a defense reaction or evidence of immunity to the cancerous growth which never quite succeeds in strangulating the malignant cells. Like fibrous tissue elsewhere, it contracts with age, often resulting in obliteration of the cavity of the gallbladder. It may produce an hour-glass deformity of the organ or squeeze stones into the common duct. The only remnant of the gallbladder may be a solid knot of scar tissue with a central calculus on the inferior surface of the liver. Should the tumor arise at the neck it may cause obstruction of the cystic duct and also empyema or mucocele of the gallbladder. In 25 per cent of the cases in the present series the growth was a scirrhous adenocarcinoma.

Mucous Adenocarcinoma.—In any of the three types mentioned, mucous degeneration may occur in one part or all of the tumor. It is a regressive change due to overgrowth of the mucous constituents of the gallbladder. Should mucous degeneration be a primary feature of the tumor, there may result extensive secretion of mucus and a bulky gelatinous mass which distends the gallbladder. Because of the friability of this tumor, there is a tendency to early perforation of the organ and extension of the lesion to the peritoneum. Twelve per cent of the cases in the present series showed isolated foci of mucous degeneration. There were 2 cases of bulky mucous adenocarcinoma.

SQUAMOUS CELL CARCINOMA

Neither during its development from the primitive gut nor in the adult form is there a vestige of squamous epithelium in the gallbladder. The occurrence of definite squamous cell carcinoma must, then, be the

result of metaplasia. This belief is borne out by the occasional presence in the mucosa of the gallbladder of local or general leukoplakia. Lubarsch [44] found a squamous cell papilloma the size of a bean. Further irritation may initiate malignant changes. A portion or all of the tumor may have squamous characteristics. Deetz [45] reported pearl formation, but keratinization is not a marked feature.[46] The gross picture is not unlike that of scirrhous adenocarcinoma. Metastases may have the same squamous characteristics or may revert to the adenocarcinomatous type. The clinical course is like that of the scirrhous or infiltrating types. It is the least common type of carcinoma of the gallbladder. But 1 example was seen in the cases at the New York Hospital.

SITE OF ORIGIN

Carcinoma may occur in any portion of the epithelium of the gallbladder. In the majority of cases (56 per cent in the present series) the growth is so extensive that it is impossible accurately to determine the origin. Cases in which the growth is less advanced show that the tumor generally arises at the fundus or in or near the neck of the organ.

It is estimated that in from 80 to 90 per cent of the cases the growth originates in the fourth of the mucosal area represented by the dome and the neck of the gallbladder. In the remaining 10 to 20 per cent it arises at some site about the lateral walls.

The explanation of this unequal distribution harks back to the etiologic rôle of calculi. Gravity and efforts of the organ to empty itself of stones cause greater irritation to the extremities than to the walls of the organ.

In the present series, the original site of the tumor could be determined in but 21 cases. In 12 of these it was at the fundus, and in 9, at or near the neck. No discrete tumors were found on the peritoneal or hepatic walls.

Obviously, the site of the tumor may influence the course of the disease and the resulting clinical picture as much as the type of tumor. A growth may exist at the fundus and not give rise to symptoms until dissemination is advanced. A scirrhous or papillary tumor at the neck, on the contrary, may cause obstruction of the cystic duct relatively

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44. Lubarsch, Otto: Pathologische Anatomie und Krebsforschung, in Arbeiten aus der pathologisch-anatomischen Abteilung des königl. hygienischen Instituts zu Posen, Wiesbaden, 1901, p. 205.

45. Deetz, E.: Vier weitere Fälle von Plattenepithel-Krebs der Gallenblase, ein Beitrag zur Frage der Epithel-Metaplasie, Virchows Arch. f. path. Anat. **164**:381-405, 1901.

46. Kodama, Shuichi: The Lymphatics of the Extrahepatic Biliary Passages, Surg., Gynec. & Obst. **43**:140, 1926.

early. A tumor on the hepatic wall of the gallbladder, by its very position, is likely to extend into or metastasize to the liver earlier than one on the free peritoneal wall, while the latter may early involve adhering peritoneal organs.

MODE OF EXTENSION

From its original site in the mucosa, a carcinoma disseminates to involve the whole gallbladder and other organs. In general, this is accomplished by local extension, lymphatic metastases and blood-borne metastases.

The lymphatics of the gallbladder consist of a rich plexus of lymph channels in the subepithelial layer and a second plexus of lymph channels in the subserosal layer, which communicate by numerous short anastomotic branches, perforating the muscular layer. The subserosal plexus in turn is collected into several main lymphatic vessels, which follow the cystic artery rather closely down onto the neck of the organ, where they terminate in the cystic node ("signal gland" of Lund). From there the lymphatics extend chiefly to the chain of mesenteric nodes which lie beside the common duct and portal vein, up to the hilus of the liver. This chain of nodes also receives branches from the pancreatic nodes, the aortic nodes and the second portion of the duodenum. Kodama [46] has demonstrated a direct lymphatic connection between the gallbladder and the first portion of the duodenum in dogs. Of great importance is the original work of Poirier and Charpy [47] and of Sudler,[48] which demonstrated an intimate connection between the lymphatics of the liver and those of the subserosal layer of the gallbladder. This work is questioned by many, but it is accepted by Graham [49] and is borne out by clinical observations.

Local extension is by far the most common method of dissemination. Infiltration first takes place through the layers of the gallbladder itself, being facilitated by the cholecystic lymphatics. Because of the arrangement of the lymphatics, the infiltrating tumor tends to encase the gallbladder on all sides early, resulting in a hard, thickened organ, which is often contracted.

The liver is involved earlier and more frequently than any other organ. This is accomplished in four ways: 1. The most common route is direct extension to the adjacent right lobe of the liver faciltated by the direct connection between the hepatic lymphatics and the subserosal

47. Poirier, P., and Charpy, A.: Traité d'anatomie humaine, Paris, Masson & Cie, 1901, vol. 4, p. 814.

48. Sudler, M. T.: The Architecture of the Gallbladder, Bull. Johns Hopkins Hosp. **12**:126-129, 1901.

49. Graham, E. A.: Diseases of the Gallbladder and Bile Ducts, Philadelphia, Lea & Febiger, 1928, pp. 30-34.

lymphatic plexus of the gallbladder. The process reduces the gallbladder and the adjacent portion of the liver to a hard knot of lobulated tumor tissue and seldom occurs until the gallbladder itself is completely infiltrated. Direct extension to the liver occurred in 66 per cent of the cases at the New York Hospital. 2. Hepatic metastases may occur through these same lymphatics. They usually are associated with direct extension, are multiple and small and on or near the surface of the right lobe of the liver in the region of the gallbladder. This type of metastases to the liver occurred in 36 per cent of the cases. In all of these there was also direct extension to the liver. The metastatic nodules varied in size between 2 and 6 cm. 3. Metastases to the liver may also occur through the periportal lymphatics in a retrograde fashion. In such cases there usually is metastatic involvement of the cystic and periportal nodes; the hepatic nodules are deep in the substance of the right lobe or rarely of the left lobe. In 1 case hepatic metastases of this type were present. There was gross involvement of the cystic and periportal nodes. 4. Recently Karlmark [50] concluded that hepatic metastases tend to localize in the quadrate lobe following the venous drainage of the gallbladder. His study concerns sarcoma as well as carcinoma of the gallbladder. Unquestionably the tumor occasionally may invade veins or arteries and thus give rise to bizarre blood-borne metastases to the liver or to distant organs.

Lymphatic metastases almost invariably involve the cystic nodes (in 52 per cent of the present series) and usually the periportal nodes (in 48 per cent of the series). Beyond the periportal nodes, lymphatic metastases are not uncommonly found in the retroperitoneal nodes about the aorta and about the pancreas (in 19 per cent of the series). The involvement of the periportal and pancreatic nodes may be massive with secondary infiltration of all surrounding structures, giving rise to obstruction of the common duct or portal vein. Occasionally, the portal vein is infiltrated by neoplastic tissue, resulting in portal obstruction or rarely in fatal hemorrhage. Infection may supervene, causing local abscess formation, pylephlebitis, thrombosis of the portal vein, multiple hepatic abscesses or septicemia.

Rarely, the mediastinal, tracheobronchial or supraclavicular nodes may be involved. Cappell and Tudhope [51] reported an unusual case with cervical metastases invading the internal jugular vein upward into the sigmoid sinus and causing hemiplegia. Strangely, there were no symptoms referable to the gallbladder.

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50. Karlmark, E.: Die Lokalisationstendenz bei Metastasierung durch die Venen in die Leber, Acta path. et microbiol. Scandinav., supp. 13, 1932, pp. 1-203.

51. Cappell, D., and Tudhope, G.: Primary Carcinoma of the Gall-Bladder with Unusual Metastases, J. Path. & Bact. **37**:167-168, 1933.

In almost every instance there is infiltration of the peritoneum of the gallbladder, but only in the late stages of the disease is the general peritoneal cavity involved. The mucous type of tumor is most likely to result in generalized peritoneal implants, in which event ascites is common.

Involvement of the omentum is probably next in frequency. This occurs by direct extension rather than by lymphatic spread, for the omentum often is adherent to the gallbladder after inflammation.

As with acute cholecystitis, so with carcinoma, the stomach, duodenum, jejunum or colon may become adherent to the gallbladder and by virtue of contiguity may be secondarily involved by tumor. Obstructive symptoms at one of these points in the gastro-intestinal tract may mask the primary symptoms of disease of the gallbladder.

The gallbladder may perforate into a neighboring viscus, causing a cholecystic fistula. Perforation into the colon occurred in 10 per cent of Musser's [7] 100 cases, while in Riedel's [52] 77 cases there was perforation into the duodenum in 10 and into the stomach in 5. Rolleston [16] considered the formation of a fistula less common in cases of carcinoma of the gallbladder than in those of cholelithiasis. In the series of cases at New York Hospital there was but 1 case of duodenal fistula, though tumor had invaded the duodenum in 5 cases, the pylorus in 2 and the hepatic flexure in 3.

Pulmonary metastases are not uncommon; they usually are very small and near the pleura in the lower lobe of the right lung. Their occurrence at this site confirms the view that they find their way from the abdomen through the retrodiaphragmatic lymphatics. Multiple and solitary metastases to other portions of the pulmonary parenchyma occur, however, and are probably blood borne. There were pulmonary metastases in 4 per cent of the series. All were in the lower lobe of the right lung.

Distant metastases are rare, but isolated case reports show that many parts of the body may be involved by secondary tumors. Beadles [53] reported an enormous metastatic tumor of the second rib. Warthin [54] discovered a case with cutaneous pigmentation due to metastases to the

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52. Riedel, B.: Zur Diagnose und Therapie des Gallenblasenkarzinomes, München. med. Wchnschr. **58**:1337-1340, 1911.

53. Beadles, C. F.: Primary Carcinoma of the Liver (Gallbladder) Associated with a Large Tumor on the Thoracic Wall, Tr. Path. Soc. London **48**:119, 1897.

54. Warthin, A. S.: A Case of Primary Adenocarcinoma of the Gall Bladder with Secondaries in Both Adrenals, Melanosis of Skin (Addison's Disease?), Vitiligo, and Hypertrophy of the Pancreas, Philadelphia M. J. **6**:38, 82 and 120, 1900.

adrenal glands. Osler [55] described a metastatic nodule in the breast. Wakasugi [56] found extensive metastatic involvement on the pelvic floor simulating carcinoma of the rectum. Rolleston [16] mentioned a case with metastatic tumor of the spine simulating tuberculosis.

CLINICAL FINDINGS

Since cholecystitis and cholelithiasis so frequently precede carcinoma, the early history in many cases is typical of inflammatory disease of the gallbladder. The first complaint may antedate the symptoms for which the patient seeks relief as long as twenty or thirty years. In other cases there are no manifestations of long-standing disease, yet in many of them gallstones are found. It is known that gallstones do not invariably cause symptoms, but this is not a likely explanation of the lack of early complaints in a large percentage of patients. A more probable reason would appear to be the relative mildness of the early symptoms.

Past History.—In only 52 per cent of the cases in the present series was there a frank history of disease of the gallbladder of long standing, yet in 70 per cent of those in which no such history was elicited stones were found. The past history was typical of intermittent biliary colic in 44 per cent of the forty-eight cases and consisted of flatulent indigestion, epigastric discomfort and intolerance of fatty foods in 8 per cent. The average duration of the past symptoms was eleven and a half years. In 1 case a single attack of painless jaundice occurred three years before the patient's admission, and in another one attack of biliary colic was followed by jaundice thirty years previously. In 3 cases cholecystostomy had been performed for acute cholecystitis associated with stones. In 1 of these cases cholecystostomy was performed three times at intervals of one year, the last operation being performed two years before the present illness. In 1 case a carcinoma developed in the stump of the cystic duct four years after a cholecystectomy for cholelithiasis.

SYMPTOMS OF CARCINOMA

After the onset of carcinoma, the clinical course of cholecystitis and cholelithiasis observed in the past history is usually modified by symptoms referable to the tumor and its growth. The symptoms attributable to carcinoma of the gallbladder are pain associated with nausea, vomiting and anorexia, followed in the course of time by loss of weight and

55. Osler, W.: On the Medical Aspects of Carcinoma of the Breast, Brit. M. J. **1**:1-4, 1906.

56. Wakasugi, K.: Zur Kenntnis der sekundären Neubildungen der Tuben, Beitr. z. path. Anat. u. z. allg. Path. **47**:483, 1910.

jaundice of the obstructive type. Some tenderness is usually elicited, and the gallbladder may be palpable as a hard mass in the right upper quadrant. These symptoms will be considered separately in the order of their frequency.

Pain.—The most common symptom in the group of cases was pain. It occurred in 43 of the 48 cases; in 69 per cent it was the first symptom of the disease. Four of the five patients without pain at the time they were seen had had attacks of biliary colic at some earlier period. The only patient in whom pain had never been a feature had an extensive tumor, which infiltrated and obstructed the duodenum.

The pain was almost invariably referred to the epigastrium or the upper right quadrant. A woman of 28 with situs inversus viscerum had pain in the left side due to carcinoma of a left-sided gallbladder. In 2 patients pain in the right upper quadrant was the only symptom.

The pain in these patients was constant, persisting over a period of time as a dull ache, with or without exacerbations of colic. In 54 per cent it was constant and dull, with an average duration of eight weeks. An additional 46 per cent suffered colicky exacerbations.

Loss of Weight.—In 95 per cent of the 48 cases loss of weight was a prominent feature. In 5 it was clearly the first symptom, but in none was it the only symptom. The loss of weight was generally dependent on the severity of the anorexia and the gastro-intestinal disturbance. The average loss of weight was 16 pounds (7.3 Kg.); the greatest loss, 100 pounds (45.4 Kg.).

Anorexia.—This symptom was present in 85 per cent of the cases. Though often severe, it was never the outstanding feature that it is prone to be in carcinoma of the stomach.

Tenderness.—Tenderness in the right upper quadrant or over the mass was found in 73 per cent of the cases. When the tenderness was marked it was associated with more or less rigidity.

Palpable Mass.—A palpable mass was present in 50 per cent of the cases. It was described as firm and tender and varied between 6 and 10 cm. in diameter. In all cases the mass proved to be a carcinomatous gallbladder. In 2 instances the discovery by the patient himself of a mass in the right upper quadrant constituted the first manifestation of the disease.

Edge of the Liver Palpable.—The edge of the liver was palpable in 50 per cent of the cases and descended between 2 and 10 cm. below the costal margin. It could be felt in the majority of patients with jaundice, though only half of these had hepatic metastases; however, some infiltration was always present. In no instance was the liver notably nodular.

Jaundice.—This symptom was present at the time of admission in 48 per cent of the cases, and in 6 it constituted the first symptom. In 4, jaundice associated with loss of weight and anorexia was the only symptom. The average icteric index was 100 and varied between 20 and 240. The average duration of the icterus was five weeks. The jaundice usually was continuous, though in 5 cases it was intermittent, and in 2 others it fluctuated. In all instances it was due not to hepatic metastases but to obstruction of the common duct. The obstruction, in turn, was caused by pressure of metastatic nodes or infiltration by the primary tumor in 91 per cent of the cases. There were but 2 cases of choledocholithiasis. Acholic stools and bile-stained urine accompanied the jaundice in most instances. Pruritus was extremely variable.

Vomiting.—According to the recent history in 45 per cent of the cases vomiting occurred at some time. In 1 it was the first manifestation of the disease. It usually appeared in mild form related to severe pain. In 2 cases, however, it was persistent, severe and the outstanding symptom; in both of these there was duodenal obstruction due to infiltration by the tumor.

Leukocytosis.—Laboratory examination showed leukocytosis to be a variable finding, occurring in 45 per cent of the cases. The average count was 14,500 and was closely related to the extent of the associated infection.

Anemia.—Anemia was not a marked feature in this group of cases. The average hemoglobin content was 74 per cent. In 2 cases of long-standing infection in the gallbladder the readings were below 60 per cent.

Chills and Fever.—These symptoms were marked in but 2 cases; obstructive jaundice was present in both, and at autopsy cholangeitis was noted. Fever at some time during the course of the disease was of extremely common occurrence.

ATYPICAL FORMS

Rarely, patients fail to present symptoms referable to the gallbladder, the first clinical manifestations of the disease being due to metastases. In this connection Lancereaux [24] described a hepatic form of the disease characterized by belching, vague abdominal pains, enlargement of the liver and slight jaundice, the clinical course of which is essentially that of carcinoma of the liver.

The clinical picture was atypical in 7 of the cases in the present series. Four of these had symptoms of obstruction of the common duct, i. e., painless jaundice with loss of weight and anorexia. In all of these metastatic nodes were obstructing the common duct. Duodenal obstruction was the dominant feature in 2 cases, in both of which vomiting was the outstanding symptom. In 1 case there was biliary colic of short

duration, with a palpable gallbladder, which is consistent with the clinical diagnosis of hydrops. At operation carcinoma was found at the fundus with a stone impacted in the cystic duct.

DIAGNOSIS

The diagnosis of carcinoma of the gallbladder is difficult to establish with certainty. With a past history of biliary colic and a recent illness characterized by steady pain, loss of weight, anorexia, obstructive jaundice and a hard palpable mass in the right upper quadrant, the diagnosis is almost certain. However, only about 30 per cent of the patients presented this combination of clinical findings.

As stated earlier, steady, dull pain in the epigastrium or the right upper quadrant proved to be the most constant symptom. The persistence of this type of pain over a period of weeks is unusual in inflammatory disease of the gallbladder. It is felt, therefore, that the type of pain is the most useful feature in differentiating carcinoma of the gallbladder and the inflammatory diseases with which it is most frequently confused. In the presence of painless jaundice, carcinoma of the common duct, ampulla of Vater or head of the pancreas must be considered. Most ampullary tumors cause occult blood in the stools, while the gallbladder is usually palpable but not tender in cancer of the head of the pancreas.

When the clinical diagnosis is uncertain, the aid of the roentgenologist usually is invoked. In spite of the recent advances in cholecystography, roentgenograms of the gallbladder seldom are of assistance in differentiating inflammatory and neoplastic diseases in this region. The reasons for this are obvious when the extent of the pathologic process in most cases of carcinoma of the gallbladder is taken into account. If sufficient normal mucosa is left to concentrate the dye and thereby render the gallbladder visible in the roentgenogram, the chances are great that calculi will obscure the picture. Often the tumor, by obstructing the cystic duct, prevents the dye from entering the gallbladder.

There are a few recent reports in the literature (notably those of Takerta,[57] Spitzenberger [58] and Albrecht [59]) which emphasize the value of the roentgenogram in diagnosing carcinoma of the gallbladder. Kirklin [60] has discussed the subject recently.

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57. Taterka, H.: Cholezytographische Untersuchungen mittels Decholindehnung und Eigelbentlehrung, Röntgenpraxis **3**:721-731, 1931.

58. Spitzenberger, O.: Zur Diagnose exulzerierender Gallenblasenkarzinome mittels des Röntgenverfahrens, Wien. klin. Wchnschr. **46**:1421-1423, 1933.

59. Albrecht, H. V.: Die Röntgendiagnostik des Verdauungskanals, Leipzig, Georg Thieme, 1931.

60. Kirklin, B. R.: Cholecystographic Diagnosis of Neoplasms of the Gallbladder, Am. J. Roentgenol. **29**:8-16, 1933.

In the present series of 48 cases, cholecystograms were made in only 17; in 9 of these the gallbladder could not be visualized, in 7 there were stones and in 1 the gallbladder was clearly outlined but failed to empty well. In these 17 cases, then, there was evidence of cholecystic disease, but in no instance could a diagnosis of carcinoma of the gallbladder be made by roentgen examination.

TREATMENT AND RESULTS

As in most malignant disease in which early diagnosis is difficult, the results of treatment are not encouraging. Operation was performed in 45 of the series of 48 cases, the various operations being exploratory laparotomy with removal of tissue for biopsy, cholecystectomy, cholecystostomy and gastro-enterostomy.

Exploration and biopsy of the tumor were carried out in 21 cases. In all of these obvious metastases were encountered, and the condition was therefore considered inoperable. Eight of the patients in this group succumbed to operation, a mortality of 38 per cent. In 5, death was due to bleeding in the presence of jaundice. The average duration of life after operation in the 13 survivors was three and one-half months.

Cholecystectomy was done in 16 cases. There were 4 postoperative deaths, a mortality of 25 per cent. In 2 of the fatal cases, the common duct was drained because of jaundice. Five patients presented no evidence of metastases at the time of operation. One of these 5 was known to be alive and well two years later but has since been lost from observation. The remaining 4 patients died, on an average of ten months after operation. There were 7 patients with known metastases who survived operation, but only for an average period of six months.

Cholecystostomy was performed on 6 patients. In all of these there was jaundice. Four died as the result of the operation, a mortality of 66 per cent. Three of the 4 deaths were due to hemorrhage. The 2 survivors died six weeks after operation.

Gastro-enterostomy was done on 2 patients as a palliative procedure for duodenal obstruction caused by tumorous infiltration. One patient died, and the other lived five weeks.

With possible cure in but 1 of 45 cases, the results of operation in this group are extremely poor. Not all authors report such discouraging experiences, however. Webber,[61] in grading Judd and Gray's[62] cases, mentioned 12 cases in which the patients lived over two years. Magoun

61. Webber, I. M.: Grades of Malignancy in Primary Carcinoma of the Gallbladder, Surg., Gynec. & Obst. **44**:756-760, 1927.

62. Judd, S. E., and Gray, H. K.: Carcinoma of the Gall-Bladder and Bile Ducts, Surg., Gynec. & Obst. **55**:308-315, 1932.

and Renshaw [63] reported 7 cases in which the patients were living five years after operation. There are occasional reports in the literature of remarkable cures [64] even after resection of adjacent, infiltrated liver.[65]

Five of the 48 patients in this series were free from metastates at the time of admission to the hospital; this confirms the contentions of Graham [25] in his excellent paper on the prevention of carcinoma of the gallbladder. He expressed the belief that the most rational attack on the disease is its prevention by removal of the gallbladder early in all cases of cholelithiasis.

Cholecystectomy has become a relatively safe procedure, and the attitude toward it is undergoing a change. Internists are more inclined to advise surgical treatment; patients are submitting to operation earlier in the course of the disease; surgeons are performing early cholecystectomy, with improved results and constantly decreasing mortality. Should this trend persist, carcinoma of the gallbladder may once more become uncommon, being confined to the cases in which the growth fails to give rise to early symptoms referable to the gallbladder.

63. Magoun, J., and Renshaw, K.: Malignant Neoplasm in the Gall-Bladder, Ann. Surg. **74**:700-720, 1921.

64. Aiga, Y.: Ueber einen seltenen Fall von operativ dauernd geheilten Gallenblasenkarzinom, Zentralbl. f. Chir. **62**:212-215, 1935.

65. Finsterer, H.: Das Karzinom der Gallenblase, Med. Klin. **28**:432-436, 1932.

PANCREATIC FISTULA

MEDICAL AND SURGICAL MANAGEMENT

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External fistula of the pancreas may be classified as either complete or incomplete. The former commonly involves the intracapitary portion of the duct of Wirsung in such manner that all the pancreatic juice is lost to the body. This event must be exceedingly rare, as the greater number of the reports encountered in the literature deal with external fistula of the incomplete variety, in which there is only a partial loss of pancreatic juice. Duodenal fistula should be differentiated, from the standpoint of classification, because that condition is complicated by the loss not only of pancreatic juice but of bile and duodenal secretion, and therefore grave and often fatal metabolic disturbances occur as a result. Furthermore, the trypsinogen of the pancreatic fluid becomes activated, and digestion of the tissues of the wound in the nearby region of the fistula rapidly ensues.

Perhaps the earliest case of pancreatic fistula recorded was that of Rommelaere in 1877.[1] Since that time numerous case reports have appeared in the literature. Fistula of the pancreas arises in various ways. It frequently follows a drainage operation on the pancreas for an acute inflammatory condition, but according to Körte,[2] it is fairly rare as a result of a stab or gun-shot wound of the abdomen. The protected anatomic location of the pancreas is the greatest safeguard against a traumatic insult of this sort,[3] but Garrè's[4] famous case of complete transverse division of the body of the pancreas with recovery, after accurate suture of the duct and parenchyma, is classic. Injury to the pancreas may follow an operation on the kidneys, spleen, stomach

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1. Rommelaere: Observation d'une fistule pancréatique chez l'homme, Bull. Acad. roy. de méd. de Belgique **9**:1023-1042, 1877.

2. Körte, W.: Die chirurgischen Krankheiten und der Verletzungen des Pankreas, in Billroth, T., and Luecke, G. A.: Deutsche Chirurgie, Stuttgart, F. Enke, 1898, no. 45, p. 212.

3. Karewski, F.: Ueber isolierte subkutane Verletzungen des Pankreas und deren Behandlung, Berl. klin. Wchnschr. **44**:187-191, 1907.

4. Garrè, C.: Totaler Querriss dess Pankreas durch Naht geheilt, Beitr. z. klin. Chir. **46**:233-340, 1905.

or duodenum.[5] In recent years several cases of pancreatic fistula have been reported after partial resection of the pancreas or excision of an islet cell tumor for the relief of hyperinsulinism.[6]

Fistula may also follow an operation for calculi of the pancreatic duct, but probably the commonest source is a cyst of the pancreas. It is seldom possible to extirpate such a cyst because of the difficulty of securing a satisfactory line of cleavage for dissection between the walls of the cyst and the adjacent organs, namely, the stomach, colon, great vessels and omentum. Marsupialization or drainage by tube is most often resorted to, and discharge of pancreatic juice for a variable period usually follows. Judd, Mattson and Mahorner,[7] in a report of thirty-three cases of pancreatic cyst treated by marsupialization, stated that the discharge continued for a period of from a few weeks up to two years. In none of their cases was further treatment necessary.

The recognition of uncomplicated pancreatic fistula is not difficult. Examination of the fluid for its reaction and enzyme content establishes the diagnosis. Duodenal fistula may be differentiated by the additional presence of bile and by the erosion of the margins of the wound.[8] Greater difficulty may be experienced in recognizing a pancreatic fistula when it is associated with a biliary fistula, as pointed out by Popper.[9]

The treatment of complete external pancreatic fistula is complicated first by the necessity of restoring pancreatic juice to the body in sufficient quantity and before serious physiologic disturbances have taken place. Hartmann and Elman [10] and McCaughan [11] have demonstrated some of the factors concerned in the mechanism of death from total pancreatic fistula in experimental animals, and McCaughan has noted the beneficial action of the daily administration of sodium chloride intravenously in prolonging the life of an animal with a fistula

5. Mayo, W. J.: The Surgery of the Pancreas, Ann. Surg. **58**:145-150, 1913. Young, H., and Davis, David M.: Young's Practice of Urology, Philadelphia, W. B. Saunders Company, 1926, vol. 2, p. 738.

6. Whipple, A. O., and Frantz, V. K.: Adenoma of Islet Cells with Hyperinsulinism: A Review, Ann. Surg. **101**:1299-1335, 1935.

7. Judd, E. S.; Mattson, H., and Mahorner, H. R.: Pancreatic Cysts: Report of Forty-Seven Cases, Arch. Surg. **22**:838-849 (May) 1931.

8. Garis, R. W., and Merkel, W. C.: The Symptom-Complex of Complete External Pancreatic Fistula: Report of a Case, Surg., Gynec. & Obst. **59**:590-597, 1934.

9. Popper, H. L.: Pankreasfermente in der Galle, Zentralbl. f. Chir. **56**: 2515-2517 (Oct. 5) 1929.

10. Hartmann, A. F., and Elman, R.: Effects of the Loss of Gastric and Pancreatic Secretions and Methods for Restoration of Normal Conditions in the Body, J. Exper. Med. **50**:387-405, 1929.

11. McCaughan, J. M.: Experimental Studies on the External Secretion of the Pancreas with Special Reference to the Effect of Its Complete Loss by Permanent Pancreatic Fistula, Am. J. Physiol. **97**:459-466, 1931.

of this type. Furthermore, the latter showed that pancreatic juice administered by gastric intubation could improve markedly the condition of an experimental animal practically moribund from the effects of long-continued total loss of pancreatic juice. The serious disturbance in the chemistry of the blood, viz., chlorides, carbon dioxide-combining power and the blood urea, were quickly restored to normal by this means. Cathala and Sénèque [12] were able to secure improvement in a patient with a pancreatic fistula by returning the pancreatic juice by intubation. It is interesting to note, however, that in animals death followed only in case of total fistula; when grave symptoms failed to develop, the fistula was shown subsequently to have been subtotal, and the animals frequently exhibited no untoward results whatever.

The treatment of pancreatic fistula is secondarily concerned with the attempts at closure of the fistulous tract. Conservative means should always be adopted in the beginning. The clinical investigations of Wohlgemuth [13] have laid the basis for this line of treatment, which consists in the administration of a diet low in carbohydrates and rich in alkaline foods and large doses of sodium bicarbonate. Belladonna, or rather atropine, was first recommended by Hartmann [13a] in 1924. According to others who later used atropine, the results were negligible. Wohlgemuth based his therapeutic regimen on the investigations of Pavlov,[14] who had noted that entrance of the acid gastric juice into the duodenum invariably provoked an increase in the flow of pancreatic juice. Wohlgemuth's measures were therefore designed to inhibit the rate of pancreatic secretion as much as possible. Heineke,[15] Hohmeier [16] and Schmidt [17] obtained satisfactory results by using Wohlgemuth's regimen in cases of their own. Heineke, in addition, advocated the aspiration of the fistula by means of a catheter and suction pump.

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12. Cathala, J., and Sénèque, J.: Fistula pancréatique: Réinjection du suc pancréatique; amélioration, pancréatico-gastrostomie, guérison, Presse méd. **38**: 1534-1537 (Nov. 12) 1930.

13. Wohlgemuth, J.: (*a*) Untersuchungen über den Pankreassaft des Menschen, Biochem. Ztschr. **2**:350-356, 1907; (*b*) Zur Therapie der Pankreasfistel nebst Bemerkungen über den Mechanismus der Pankrassekretion während der Verdauung, Berl. klin. Wchnschr. **45**:389-393, 1908; (*c*) Beitrag zur funktionellen Diagnostik des Pankreas, ibid. **47**:92-95, 1910.

13a. Hartmann, M., in discussion on Barthélemy, M.: Pancréatite aiguë sans stéatonécrose, Bull. et mém. Soc. nat. de chir. **50**:408, 1924.

14. Pavlov, I. P.: The Work of the Digestive Glands, translated by W. H. Thompson, ed. 2, London, C. Griffin & Co., 1910, p. 266.

15. Heineke, H.: Zur Behandlung der Pankreasfisteln, Zentralbl. f. Chir. **34**: 265-296, 1907.

16. Hohmeier, F.: Isolierte subkutane Querzerreissung des Pankreas durch Operation geheilt, München. med. Wchnschr. **54**:2036-2037, 1907.

17. Schmidt, W.: Ein Fall von Totalexstirpation einer Pankreaszyste, München med. Wchnschr. **54**:2480-2482, 1907.

Kroiss [18] recommended the administration in 50 Gm. doses, by mouth or by rectum, of a water-free product prepared by the digestion of meats and consisting largely of the amino-acids thus produced. (The specific product employed [erepton] is no longer on the market.) Ortlich [19] injected astringents and corrosives, such as tincture of iodine, silver nitrate and zinc chloride. Potter [20] applied tenth-normal hydrochloric acid to the fistulous tract by means of a dropper and between times maintained pledgets of gauze soaked in a solution of peptone in the cutaneous opening of the fistula. The use of high voltage roentgen therapy was reported as successful by Culler [21] in the cases of two soldiers with pancreatic fistula. In each case the fistula was said to have closed completely after five irradiations. Orndoff, Farrell and Ivy,[22] on the other hand, studied the effects of roentgen therapy on the pancreas in laboratory animals and concluded that while the pancreas could be temporarily injured, its power of regeneration later on overcompensated, and the total output of ferments might become even greater than normal. Our own experience in giving high voltage roentgen therapy to a patient with a pancreatic fistula and a cyst following partial pancreatectomy for hyperinsulinism (to be reported elsewhere) is in accord with the conclusions of Orndoff, Farrell and Ivy. We exposed the left upper quadrant of the abdomen to a current of 200 kilowatts and 5 milliamperes for fifteen minutes at a time, giving a total exposure of seventy-five minutes. There was no noticeable improvement, and surgical incision and drainage later became imperative.

If these conservative methods fail after trial for a reasonable period, radical measures should then be considered. The excision or extirpation of the entire fistulous tract for obvious reasons is rarely possible. In 1905 Doyen [23] reported the first instances of successful implantation of the pancreatic duct into the greater curvature of the stomach after resection of the papilla of Vater for carcinoma. Kausch [24] in 1909 reported the successful implantation of the stump of the pancreas into the duodenum after resection of a carcinoma of the papilla of Vater.

18. Kroiss, F.: Ein Beitrag zur Behandlung der subkutanen Duodenum-und Pankreaszerreissung, Beitr. z. klin. Chir. **76**:477-495, 1911.

19. Ortlich, cited by Kleinschmidt.[31]

20. Potter, C.: Treatment of Duodenal, High Intestinal and Pancreatic Fistulas, J. Missouri M. A. **29**:374-378, 1932.

21. Culler, R. M.: Cure of Pancreatic Fistula by the Roentgen Ray, J. A. M. A. **75**:20 (July 3) 1920.

22. Orndoff, B. H.; Farrell, J. I., and Ivy, A. C.: Studies on the Effect of Roentgen Rays on Glandular Activity: V. The Effect of Roentgen Rays on External Pancreatic Secretion, Am. J. Roentgenol. **16**:349-354, 1926.

23. Doyen, cited by Kleinschmidt.[31]

24. Kausch, W.: Die Resektion des mittleren Duodenum: Eine typische Operation, Zentralbl. f. Chir. **36**:1350-1376, 1909.

The experimental work of Coffey,[25] Desjardins [26] and von Fáykiss [27] on pancreatico-enterostomy was an important forward step in this difficult field of surgical technic. After the work of Doyen and Kausch on the transplantation of the pancreatic duct after resection of the papilla of Vater for carcinoma, others began to utilize the principle of this method in dealing with intractable fistula of the pancreas, particularly Jedlicka [28] (1921), Lorenz [29] (1921), Hammesfahr [30] (1923), Kleinschmidt [31] (1925), Corachan [32] (1924, Cathala and Sénèque [12] (1930) and Janes [33] (1934).

The technic consists in making a careful dissection of the fibrous fistulous tract until a sufficient length has been mobilized. This may then be implanted without tension into the nearest portion of the upper part of the gastro-intestinal tract, preferably the stomach. A tag of fat from the round ligament or the gastrocolic or gastrohepatic omentum or even a free graft of fat is stitched about the site of implantation to protect the line of suture against leakage. A small drainage tube is placed near the site of operation, and the abdominal wound is closed in the usual manner.

The entrance of pancreatic juice into the stomach seems to have no deleterious effect on gastric digestion, according to Grey [34] and Tripodi and Sherwin,[35] who investigated this problem in dogs. Ascending infection by way of the duct, even when the implantation was made into the descending portion of the colon of dogs, did not take place. In Kausch's case of pancreaticoduodenostomy for carcinoma of the papilla of Vater clinical evidence of freedom from ascending pancreatic infection is afforded. The patient died one year after operation, and the

25. Coffey, R. C.: Pancreato-Enterostomy and Pancreatectomy, Ann. Surg. **50**: 1238-1264, 1909.

26. Desjardins, cited by Kleinschmidt.[31]

27. von Fáykiss, F.: Ueber experimentelle Pankreasresektion und Pankreatoenterostomie, Beitr. z. klin. Chir. **84**:188-200, 1913.

28. Jedlicka, R.: Zur Operation der Pankreas Kysten, Zentralorg. f. d. ges. Chir. **16**:153, 1922.

29. Lorenz, H.: Kasuistische Beiträge Zur Pankreas-und zur Gallenchirurgie, Wien. klin. Wchnschr. **34**:339-340, 1921.

30. Hammesfahr, C.: Zur Behandlung von Pankreasfisteln, Zentralbl. f. Chir. **50**:1758-1759, 1923.

31. Kleinschmidt, O.: Behandlung der Fisteln des Pankreas und des Ductus pancreaticus, Arch. f. klin. Chir. **135**:363-372, 1925.

32. Corachan, M.: Sur le traitement des fistules pancréatiques, Presse méd. **36**:1394-1397 (Nov. 3) 1928.

33. Janes, R. M.: Pancreatic Fistula: Report of a Case; Cure by Pancreato-Gastrostomy, Brit. J. Surg. **22**:296-300, 1934.

34. Grey, E. G.: Diversion of the Pancreatic Juice, J. Exper. Med. **26**:825-839, 1917.

35. Tripodi, A. M., and Sherwin, C. J.: Experimental Transplantation of the Pancreas into the Stomach, Arch. Surg. **28**:345-356 (Feb.) 1934.

pancreas appeared to be quite normal at necropsy. The mucous membrane of the duodenum had covered the stump of the pancreas up to the opening of the duct, which was about the size of a goose-quill.

The following is a report of a case of pancreatic fistula in which the fistula was transplanted into the stomach.

REPORT OF A CASE

A. F., a white man 49 years of age, a molder, was admitted to the Firmin Desloge Hospital on Jan. 15, 1936. He was referred to one of us (McCaughan) for the surgical closure of a pancreatic fistula, which had followed a Billroth II type of gastric resection. The history is as follows: Nine months prior to the present admission to the hospital the patient was admitted to another institution, complaining of a dull aching pain in the epigastrium of two years' duration. By January 1935 the pain, which came on rather promptly after meals, had become severe, and was accompanied by nausea and vomiting. There had been a loss of 35 pounds (15.9 Kg.) in weight during the preceding twelve months. The nonprotein nitrogen content of the blood was 85.7 mg. per hundred cubic centimeters. Roentgenographic examination of the gastro-intestinal tract at that time revealed an enormously dilated stomach with marked retention and an apparent deformity of the prepyloric segment, which was interpreted as a gastric neoplasm. On March 30 an exploratory operation was performed on the stomach, and a firm thick mass about the size of a hen's egg was found occupying the prepyloric segment of the stomach. There were no palpable lymph glands, but numerous adhesions about the pylorus, duodenum and pancreas were encountered. A posterior gastro-enterostomy was done, and resection was deferred until later because of the poor condition of the patient. On April 15, two weeks afterward, the abdomen was reopened, and the mass in the prepyloric segment of the stomach was found to have shrunk considerably in size. A partial duodenectomy and gastrectomy were done. About one third of the stomach was removed. On the fifth day after operation the patient went into moderate shock, but he rallied after a transfusion of blood. On the eighth day postoperatively a copious watery discharge came from the wound. The patient improved generally thereafter, but the discharge persisted. The flow was practically continuous, but there was a marked increase after meals and a considerable diminution during fasting and at night. On several occasions drainage ceased for periods of from one to five days, but in two such instances bits of silk suture material were extruded from the fistula, and this was followed promptly by the return of drainage. The Wohlgemuth treatment was instituted in an endeavor to bring about healing of the fistula by medical methods, but the results were unsatisfactory. At the time of entry into the Firmin Desloge Hospital the patient stated that the fistula had been draining for more than nine months.

On physical examination the patient appeared markedly undernourished. The teeth were very bad and exhibited a great many caries. Pyorrhea alveolaris was present. There were a few coarse râles in the left side of the upper part of the chest posteriorly, but otherwise the thorax was normal. There was an old median operative scar in the upper part of the abdomen, and about 4 cm. below the xyphoid process there was a tiny cutaneous opening which barely admitted the tip of a small probe. The surrounding skin appeared to be normal. A clear watery fluid was flowing profusely from this opening (fig. 1). A sample of the material was collected for study. The fluid was strongly alkaline. It had a p_H of 8.56, and all three of the pancreatic enzymes could be demonstrated. A bacteriologic examination of the fluid showed gram-positive cocci in clusters and gram-

negative rods which fermented lactose. These were probably contaminators from the skin, as neither fistula nor fluid presented any gross evidence of infection. The systolic blood pressure was 125 mg. of mercury and the diastolic, 94 mm. The pulse rate was 72 and the temperature 98 F. The body weight at the time of admission was 118 pounds (53.5 Kg.).

On laboratory examination the urine was normal, except for an occasional leukocyte and erythrocyte microscopically. The blood count showed 7,900 leukocytes and 3,940,000 erythrocytes; the hemoglobin content was 13 Gm. per hundred cubic centimeters. The differential count was normal. The clotting and bleeding time were both normal, as was the clot retraction time. Studies were made of the blood chemistry, with the following results: sugar, 151 mg. per hundred cubic centimeters; nonprotein nitrogen, 33 mg.; carbon dioxide content, 49 volumes per cent, and chlorides, 590 mg. A sugar tolerance test by the Shaffer-Hartmann method gave a curve within the normal zone. The Wassermann and Kahn tests of the blood were negative. Examination of the sputum gave negative results. A gastric analysis showed normal free and combined acids. Roentgenograms

Fig. 1.—External fistula of the pancreas, indicated by the pointer.

were taken of the chest, and a fluoroscopic examination of the stomach was made. The roentgenographic examination revealed a moderate degree of peribronchial thickening and emphysema at the bases of both lungs. The interlobular pleura on the right was thickened. A diagnosis of chronic bronchitis was made. The fluoroscopic examination showed a normal esophagus, but the stomach had been resected at its distal third. A gastro-enterostomy stoma was evident 2 inches (5 cm.) proximal to the blind end of the stomach. Barium sulfate passed freely through the stoma.

Physiologic studies were made during the next two weeks, and the responses to various excitatory and inhibitory drugs and foodstuffs were determined. These results will be reported elsewhere. On two occasions prior to operation there was a temporary cessation of drainage, followed by headache, nausea, general malaise and an elevation of temperature to 102 F. These symptoms cleared up quickly with the return of drainage. No reason could be advanced to explain this apparent retention of pancreatic juice.

On Feb. 1, 1936, a transfusion of 600 cc. of citrated blood was given, and on February 10, with the patient under spinal anesthesia, an exploratory operation was performed on the abdomen. The incision was made at the site of the second operative scar, and a small area of skin about the fistula was left in situ. A probe was introduced into the tract as a guide, and the fistula with its attached island of

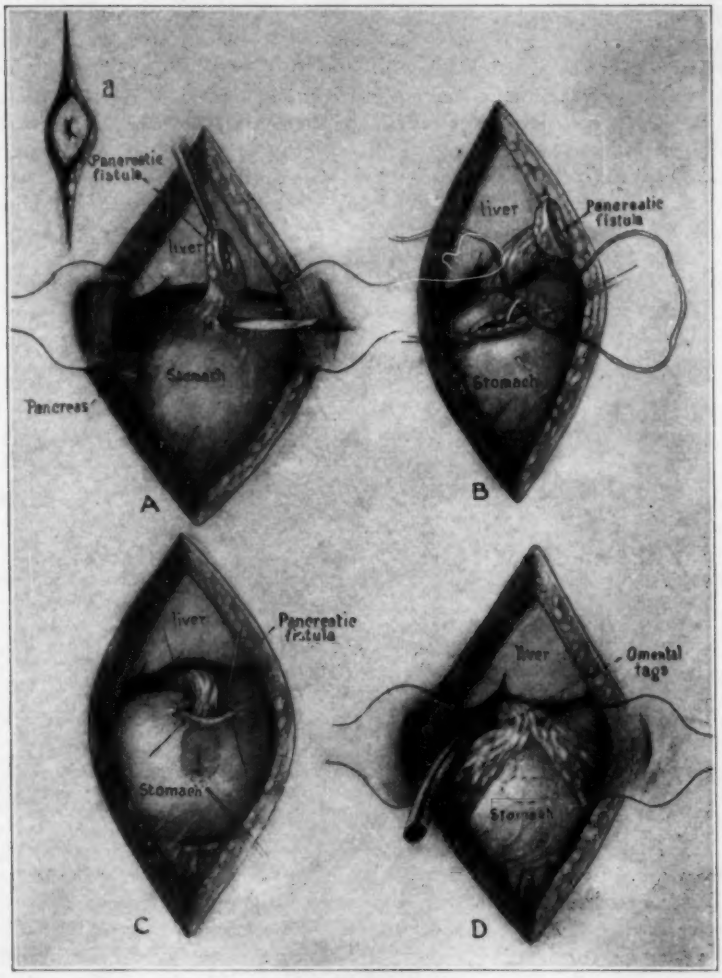

Fig. 2.—The operative technic used in the case reported here. The incision is shown in the small insert (*a*). An island of skin surrounding the opening of the fistula was left intact. In *A*, the fistula is being dissected downward. The cicatricial attachment to the lesser curvature and posterior wall of the stomach is being divided by sharp dissection, to obtain complete mobilization. The head of the pancreas is visible in the lower left side of the wound. In *B*, the stomach has been opened, and the fistula is being prepared for implantation and temporary fixation until gastric closure can be completed. In *C*, the wall of the stomach is being sutured about the fistula. In this procedure care should be taken not to constrict the tract or to produce angulation. In *D*, the omental tags of fat are shown stitched about the site of implantation, and the traction suture has been withdrawn, permitting the distal end of the fistula to hang downward. The cuff of skin prevents the fistula from slipping out. A small drain is introduced alongside the site of implantation before the abdominal closure is made.

skin was dissected free and a segment 5 cm. in length mobilized. The fistula measured approximately 1 cm. in diameter. The walls of the tract were rather thin, and some difficulty was encountered in freeing it from the lesser curvature and posterior wall of the stomach. This was accomplished by sharp dissection without injury to the stomach or to the fistula. The adhesions from the two previous laparotomies had walled off the operative field in such a manner as to separate it from the rest of the peritoneal cavity. The fistula was then implanted into the anterior wall of the stomach near the blind end. The technic employed is shown in figure 2. After operation the patient did very well until the fourth day, when a purulent expectoration and a temperature of 104 F. developed. Physical examination of the chest revealed a few moist râles. A roentgenogram showed nothing except a moderate degree of peribronchial thickening as before. These symptoms cleared up rapidly under appropriate treatment. There was

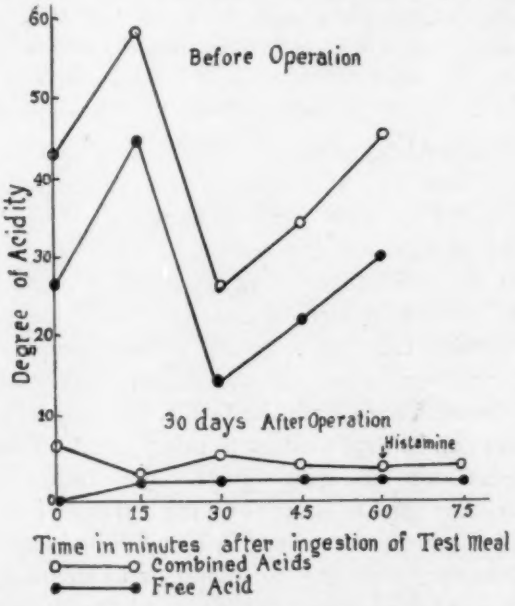

Fig. 3.—Curves showing the degree of gastric acidity before and four weeks after the implantation of the pancreatic fistula into the stomach. The test meal consisted of 240 cc. of 8 per cent ethyl alcohol given by intubation immediately after the removal of the specimen during fasting. In the postoperative analysis, 1 cc. of histamine was given after the fourth, quarter hour interval.

considerable drainage from the wound for the first seventy-two hours. The physical and chemical characteristics of this drainage were identical with those of the fluid from the fistula which was observed previous to operation. At the same time the suture material (no. 2 chromic catgut) gave way apparently as a result of the digestive action of the escaping fluid, and the wound separated completely for a distance of about 4 cm. transversely and about 6 cm. vertically. The base of the wound was seen to be formed by the liver superiorly and the anterior wall of the stomach inferiorly and, covering both, the mesenteric fat which had been drawn up at the time of operation to protect the implanted fistula. Through and through sutures of silkworm gut above and below, however, prevented complete dehiscence. A purulent exudate appeared later, but the leakage

of pancreatic juice ceased abruptly, and with the constant application of gauze soaked in allantoin the infection cleared up gradually, and a growth of healthy granulations soon filled the defect. At the end of four weeks the wound was entirely healed except for a small crust at its center. The patient rapidly regained his weight and strength and felt much improved. Samples of the gastric fluid were also examined for lipase and were found to possess definite lipolytic activity. An index of 0.3 was found when the index for duodenal lipolytic activity by the method employed was from 1.5 to 2 units. A gastric analysis was made for comparison with that obtained before operation, and the degree of acidity was found to have fallen considerably (fig. 3). The diastase in the blood was estimated quantitatively and found to be within normal limits.

The patient was discharged from the hospital on March 15. Two months after operation we were advised that he was gaining in weight and strength. His appetite was fair. There was no dyspepsia and no diarrhea. There was no discharge from the wound, but a small herniation had developed in the lower angle of the wound. This was controlled satisfactorily with a belt.

COMMENT

A case of incomplete pancreatic fistula in which the implantation of the fistulous tract into the stomach was successful, after medical measures had failed to effect healing, is reported. The literature on pancreatic fistula is reviewed, and both conservative and radical methods of dealing with incomplete fistula are discussed in detail. It is difficult to state definitely how long medical measures should be employed before one resorts to surgical treatment. In this instance medical treatment was carried out for perhaps eight or nine months without any significant diminution in the discharge from the fistula. It is questionable whether these procedures can be depended on to bring about a continuous inhibition in the secretion of pancreatic juice. We studied the effect of a variety of drugs and food substances on the activity of pancreatic secretion in this patient, and our results will be published elsewhere. Suffice it to say here that we were able to inhibit or to excite the flow with a number of the substances given, but we question whether these effects could be effectively maintained over long periods of time. Certainly in cases in which there is a well defined fistulous tract with possibly a cicatricial stenosis proximal to it, it would appear futile to continue with the medical regimen. High voltage roentgen therapy had proved unsuccessful in a previous case (to be published elsewhere) and so was not again attempted.

The success of any surgical technic for transplantation will depend on obtaining free mobilization of the fistulous tract and its implantation without tension into the nearest available portion of the upper part of the gastro-intestinal tract, preferably the stomach. It is essential that there are no constrictions or acute angulations, since retention would thereby be favored and the development of a cyst practically a certainty. The tract should not be stitched to the wall of the stomach but should be allowed to find its own proper degree of tension exactly as in the

technic for the production of a perineal urethra. If a suitable island of skin with the fistula opening at the center is left attached, the tract is not likely to slip out, and ultimately that portion beyond the site of union between the intestine and the tract may be expected to slough away as a result of autodigestion. Temporary leakage of gastric and pancreatic juice should be expected, but if the tract is patent it should cease within a few days. The final step in the operative procedure, namely, the utilization of the tags of fat from the adjacent omentum, is an important factor in minimizing the danger of permanent leakage.

The influence on gastric acidity after transplantation is of especial interest. Grey,[34] in a series of experiments, transplanted the major pancreatic duct into the wall of the stomach in dogs in which previously a Janeway fistula had been produced. Using a test meal of 70 Gm. of beef and 75 cc. of tap water, he withdrew samples from the fistula at intervals. His results in the main were in agreement with the work of Boldyreff on the self-regulation of the acidity of the stomach and illustrated a remarkable compensatory activity of the gastric glands. He found that the presence of considerable amounts of pancreatic juice in the stomach during digestion led only to a moderate decrease in the acidity level of the ingesta. After months, Grey could find no evidence of inflammatory or degenerative change in the pancreatic glands, although the patency of the ducts could be demonstrated in every instance. The marked decrease in acidity which obtained in our patient differs greatly from Grey's observation on his animals and may be the result of a difference in species. It is more likely due, however, to the fact that the principal acid-forming area of the stomach had been previously extirpated, and the remaining portion was unable to compensate in a normal manner.

In a recent review of the literature on hyperinsulinism, Whipple and his co-workers [6] reported the occurrence of pancreatic fistula in three of thirty-five cases in which operation was performed. In a similar study, we concluded that pancreatic fistula is more likely to follow the local excision of a pancreatic adenoma than partial resection of the pancreas, in which transverse division and complete removal of the distal portion are practiced. This conclusion received experimental support from the work of Korovitsky,[36] who showed that the ducts were not mere conducting tubes as some supposed, but that they possessed a definite tonus which propelled the pancreatic juice toward the duodenum.

The possibility of inadvertent and even unpreventable trauma to the pancreas during certain intra-abdominal operations should be kept in mind. Such injuries may go unrecognized at the time, but if the surgeon is aware of having injured the pancreas, there is the choice of several

36. Korovitsky, L. K.: The Part Played by the Ducts in Pancreatic Secretion, J. Physiol. **57**:214-233, 1923.

procedures, depending, of course, on the location and extent of the wound in the pancreas. In superficial injuries the wound may be closed with a few interrupted sutures and a small drain introduced to the site of the injury before the abdomen is closed. In the case of a more extensive injury the surgeon may elect to do nothing and adopt a policy of watchful waiting with an idea of dealing with complications if and when they occur, or in cases in which the injury is located in the body or tail, he may attempt to circumvent trouble by removing the involved segment and the distal portion of the pancreas. If the major pancreatic duct were divided in the head or in the body, for example, one might attempt to resuture it, as in Garrič's classic case. Perhaps the simplest step of all with injuries situated in the body or tail would be to ligate the pancreas securely at the site of injury. The ensuing atrophy of the acinous cells proceeds rapidly without evident clinical disturbance in experimental animals in which the pancreatic ducts have been divided between ligatures. The islet tissue remains intact, and no interference with carbohydrate metabolism occurs. The digestive disturbances which follow insufficiency of the external pancreatic secretion are the result of the excessive loss of fat and nitrogen in the stools. This loss can now be fairly well controlled by the administration of certain vegetable diastases.[37] Drainage should be instituted before closure of the abdomen, in any event, particularly if nothing else can be done at the time, as the avoidance of retention and subsequent pancreatic cyst formation would seem to be especially desirable.

The likeliest sources of danger in abdominal operations are, in the order named: operations on the stomach and duodenum, operations on the biliary tract, operations on the spleen, operations on the kidneys, operations for retroperitoneal tumor and operations on the adrenal glands.

CONCLUSION

External fistula of the pancreas may be complete or incomplete. The incomplete type is the more common as well as the least serious. The treatment of incomplete pancreatic fistula is at first medical, and the basis of such management has been discussed.

The surgical treatment is indicated when conservative measures fail after a reasonable period to bring about definite and continued improvement. The surgical treatment consists in the implantation of the fistula in some readily accessible portion of the upper part of the gastrointestinal tract.

A case of an incomplete pancreatic fistula in which successful transplantation of the fistula into the stomach was done is reported in detail, and the literature is reviewed.

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37. Whipple, A. O.; Parsons, W. B., and Mullins, C. R.: Treatment of Carcinoma of the Ampulla of Vater, Ann. Surg. **102**:763-779, 1935.

PHYSIOLOGIC AVAILABILITY OF FLUIDS IN SECONDARY SHOCK

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The central factor in the production of traumatic shock is the reduction of the volume of blood. The exact mechanism of the oligemia occurring in shock is debatable, but at the present time most consideration has been given to three hypotheses. According to the first, vasotropic substances are formed in the area of injury and are absorbed into the general circulation, producing a marked fall in the blood pressure and an increase in the permeability of the capillaries. As a result, blood and blood plasma pass out into the extravascular tissues, and a reduction in the volume of blood takes place (Cannon,[1] Cannon and Bayliss [2] and Grunke and Haring [3]).

Subsequent observers, however, have been unable to obtain convincing evidence of the presence of such toxic products either in the area of injury (Blalock,[4] Parsons and Phemister [5] and Simonart [6]) or in the blood stream of human beings suffering from shock (Schneider [7] and Herbst [8]).

According to the second hypothesis, the reduction in the volume of blood may be satisfactorily accounted for by a loss of plasma and blood

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1. Cannon, W. B.: Experimental Traumatic Shock: Evidence of a Toxic Factor in Wound Shock, Arch. Surg. **4**:1 (Jan.) 1922.

2. Cannon, W. B., and Bayliss, W. M.: Note on Muscle Injury in Relation to Shock, Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions, Medical Research Committee, London, His Majesty's Stationery Office, 1919, p. 19.

3. Grunke, W., and Haring, W.: Das Verhalten der Blutmenge beim experimentellen Shock, Ztschr. f. d. ges. exper. Med. **79**:763, 1931.

4. Blalock, A.: Experimental Shock: Cause of Low Blood Pressure Produced by Muscle Injury, Arch. Surg. **20**:959 (June) 1930.

5. Parsons, E., and Phemister, D. B.: Haemorrhage and "Shock" in Traumatized Limbs: Experimental Study, Surg., Gynec. & Obst. **51**:196, 1930.

6. Simonart, A.: Etude expérimentale sur la toxémie traumatique et la toxémie des grands brûlés, Arch. internat. de pharmacodyn. et de thérap. **37**:269, 1930.

7. Schneider, H.: Ueber das Vorkommen und über die Bedeutung von Gewebsgiften bei Shockzuständen des Menschen, Deutsche Ztschr. f. Chir. **229**: 343, 1930.

8. Herbst, R.: Experimentelles zur Entstehung des traumatischen Shocks, Arch. f. klin. Chir. **176**:98, 1933.

into the traumatized region (Blalock,[4] Parsons and Phemister[5] and Herbst[8]). The basis of the third point of view is that a prolonged overactivity of the sympathetic nervous system is capable per se of reducing the volume of blood to a shock level (Freeman[9] and Malcolm[10]). This conclusion followed the observation of a diminished volume of blood in decorticate animals exhibiting sham rage and after prolonged injections of epinephrine. In both instances a hyperactivity of the sympathetic nervous system was readily demonstrable.

Whatever may be the mode of its production, the oligemia causes widespread physiologic effects, in particular an oxygen want which is directly proportional to the reduction in the volume of blood. Even a small hemorrhage and minor grade of shock induces a considerable diminution in the volume flow of blood to peripheral organs, amounting in certain instances to a decrement of as much as 80 per cent (Gesell[11]). The administration of fluid increases the flow of blood through peripheral blood vessels, even though the fluid possesses no oxygen-carrying properties. However, as will be shown later, an increased volume flow to the periphery does not result, of necessity, in an increased utilization of oxygen by anoxemic tissues.

FLUIDS IN SHOCK

In evaluating the fluid therapy of shock, two questions arise: 1. How adequate are fluids in the treatment of shock? 2. Which criteria can be considered most significant in the estimation of the depth of shock? The adequacy of a fluid in the treatment of shock must depend primarily on its ability to produce and maintain an increased volume of blood. Judged by this standard, blood is the most effective fluid available. The beneficial results obtained by the use of a 6 per cent solution of acacia (Bayliss[12]) led to its wide adoption as a therapeutic measure in cases of shock. Solution of acacia itself is incapable of transporting oxygen, but in view of the fact that in shock there is relatively little diminution of the oxygen-carrying erythrocytes in the blood stream and that concentration of the red blood cells almost invariably occurs, the use of this drug in the treatment of shock rests on a reasonable basis. Hypertonic solution of acacia and dextrose solution have been advocated

9. Freeman, N. E.: Decrease in Blood Volume After Prolonged Hyperactivity of Sympathetic Nervous System, Am. J. Physiol. **103**:185, 1933.

10. Malcolm, J. D.: The Nature and Treatment of Surgical Shock, Tr. Med. Soc. London **32**:274, 1909.

11. Gesell, R.: Studies on Submaxillary Gland: I. Dependence of Tissue Activity upon Volume-Flow of Blood and on Mechanism Controlling This Volume-Flow of Blood, Am. J. Physiol. **54**:166, 1920.

12. Bayliss, W. M.: Intravenous Injection in Wound Shock, London, Longmans, Green & Co., 1919.

(Erlanger and Gasser [13]), but the danger of acute cardiac dilatation after their administration has prevented their general use. The ready availability of isotonic solutions of sodium chloride and dextrose solutions has made them the commonly used fluids in the treatment of shock and hemorrhage. During an investigation on the metabolic effects of water, a peculiar abnormal response was noticed in animals which had been subjected to hemorrhage (Davis [14]).

This investigation was undertaken to determine the nature and mechanism of the response of normal animals and of animals in shock to water and electrolytes. The question was raised regarding the criteria of shock to be used in this study. In view of the fact that the central phenomenon of shock is an anoxemia which is the resultant of such factors as oligemia, lowered blood pressure, diminished volume flow of blood and peripheral vasoconstriction, it was decided that the rate of oxygen consumption of the animal provided a more accurate picture of the effects of traumatic shock on the tissues. However, determinations of the blood pressure level were made to serve as controls.

METHODS

Healthy dogs weighing from 10 to 20 Kg. were used throughout these experiments. Anesthesia was induced by sodium barbital in a dosage of 250 mg. per kilogram of body weight. Surgical anesthesia was essential in order to prevent movements which might falsify the metabolism readings. The metabolic rates of several unanesthetized animals, which had been trained for such determinations, were obtained. Sodium barbital was thereafter administered in the aforementioned dosage, and only a slight depression of the rate of oxygen consumption was noticed. Moreover, the metabolism readings of trained unanesthetized animals often showed variations of from 100 to 300 cc. of oxygen consumption per ten minutes. Especially was this noticeable when readings were obtained frequently and over long periods of time. In the animals anesthetized with sodium barbital, the rate of metabolism remained remarkably constant. The metabolism determinations were made with a Krogh respiratory apparatus before and at frequent intervals during each experiment. Prior to each experiment the urinary bladder was emptied by means of a catheter, which was left in situ, in order that the urinary secretion might be measured. Fluid was administered in the form of an isotonic solution of sodium chloride (0.9 per cent) heated to 38 C. This was permitted to run into the exposed femoral vein at a constant rate of 16 cc. per minute until 3,000 cc. was given, at which point the injection was stopped. A control series of twenty normal dogs was used. In another series of fifteen animals secondary shock was produced by (1) massive hemorrhage to the extent of one third of the calculated volume of blood, (2) repeated trauma to a hindlimb, (3) ligation of the portal vein and (4) the injection of histamine dihydrochloride.

13. Erlanger, J., and Gasser, H. S.: Hypertonic Gum Acacia and Glucose in the Treatment of Secondary Traumatic Shock, Ann. Surg. **69**:389, 1919.

14. Davis, H. A.: The Relation of Water and Electrolytes to Metabolism, Science **81**:493, 1935.

The blood pressure was recorded in the usual manner in the majority of these experiments.

Death following ligation of the portal vein has been ascribed to shock resulting from loss of blood and plasma into the wall of the bowel (Elman and Cole[15]). In this investigation it has been found that the onset of shock after obstruction of the portal vein may be appreciably hastened by the intravenous injection of an isotonic saline solution. Postmortem examination of these animals revealed an intense edema of the walls of the large and small intestines. The spleen was enlarged to as much as three times its normal size.

FLUIDS AND METABOLISM

The metabolic effects of water have been investigated both in human beings and in experimental animals, and it has been shown that the administration of water increases the rate of metabolism (Eichhorst,[16] Feder,[17] Falck and Falck,[18] Genth,[19] Gruzdiev,[20] Matzkevich,[21] Becker,[22] Neumann,[23] Panum,[24] Schöndorff[25] and Weigelin.[26] However, the most direct evidence that an increased intake of fluid augments cellular activity was provided by Hawk and his co-workers,[27] when they demonstrated

15. Elman, R., and Cole, W. H.: Hemorrhage and Shock as Causes of Death Following Acute Portal Obstruction, Arch. Surg. **28**:1166 (June) 1934.

16. Eichhorst, H.: Ueber die Resorption der Albuminate im Dickdarm, Arch. f. Physiol. **4**:570, 1871.

17. Feder, L.: Der zeitliche Ablauf der Zersetzung im Thierkörper, Ztschr. f. Biol. **17**:531, 1881.

18. Falck, E. P., and Falck, F. A.: Physiologische Studien über die Ausleerungen des auf absolute Carenz gesetzten Hundes, Beitr. z. Physiol., Hyg. u. Toxicol. **1**:1, 1875.

19. Genth, E. A.: Untersuchungen über den Einfluss des Wassertrinkens auf den Stoffwechsel, Wiesbaden, Kreidel & Niedner, 1856.

20. Gruzdiev, V. S.: Comparative Influence of Copious or Moderate Drinking of Water on Change of Matter, Vrach **11**:213, 1890.

21. Matzkevich, V. G.: Influence of Abundant Water Drinking on Assimilation of Nitrogenous Particles of Food, and Nitrogenous Metabolism in Typhoid Patients, St. Petersburg, P. Voshtshinskaya, 1890.

22. Becker, N. M.: De l'influence des solutions de bicarbonate de soude, de sel marin, d'acide carbonique et de quelques eaux alcalines sur la sécrétion du suc pancréatique, Arch. d. sc. biol. **2**:433, 1893.

23. Neumann, R. O.: Die Bedeutung des Alkohols als Nahrungsmittel, Arch. f. Hyg. **36**:1, 1899.

24. Panum, P. L.: Om urinstof—og urinsekretionens kurve efter en enkel maaltid om dagen, bestaaende af kød med eller uden tilsaetning af fedt, borsyre, rugbrud og vand, Nord. med. ark. **6**:1, 1874.

25. Schöndorff, B.: Ueber den Einfluss des Wassertrinkens auf die Ausscheidung der Harnsäure, Arch. f. Physiol. **46**:601, 1890.

26. Weigelin, J.: Versuche über die Harnstoffausscheidung während und nach Muskelthätigkeit, Arch. f. Anat. u. Physiol. **3**:207, 1868.

27. Howe, P. E.; Mattill, H. A., and Hawk, P. B.: The Influence of an Excessive Water Ingestion on a Dog After a Prolonged Fast, J. Biol. Chem. **10**:417, 1911-1912.

an increased excretion of total purine and nitrogen after the administration of fluid. The stimulating effect of water on metabolism which was demonstrated by these earlier workers was fully corroborated by subsequent investigators (Jarisch and Liljestrand,[28] Carpenter and Fox,[29] Rubner,[30] Barcroft,[31] Zuntz,[32] Verzár,[33] Tangl,[34] Janssen and Rein,[35] Lublin [36] and Grollman [37]). The degree of stimulation produced by water is largely dependent on the mode of administration. When fluid is given subcutaneously, the rise in metabolism may be absent completely. A somewhat greater response results from the oral administration of water, and it has been pointed out that the ingestion of 500 cc. of water may increase the metabolic rate in man as much as 16 per cent above the initial level (Benedict and Carpenter [38]). The intravenous ingestion of fluid causes a considerable rise in the rate of oxygen consumption. In the initial control experiments the following solutions were used: 0.9 per cent solution of sodium chloride, 5 per cent solution of dextrose and 10 per cent solution of sucrose. In each case a rise in metabolism occurred, the highest rates being encountered when dextrose solution was administered. In some instances the metabolic rate was increased by 200 to 300 per cent when dextrose solution was given, but the rise was not sustained and soon returned to the original level. Saline and sucrose solutions did not produce such great augmentations of the metabolism, which varied from 25 to 50 per cent, but the increase was longer sustained.

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28. Jarisch, A., and Liljestrand, G.: Ueber das Verhalten des Kreislaufes bei Muskelarbeit nach dem Essen und bei Flüssigkeitszufuhr, Skandinav. Arch. f. Physiol. **51**:235, 1927.

29. Carpenter, T. M., and Fox, E. L.: The Gaseous Exchange of the Human Subject as Affected by Ingestion of Water at 37° C., J. Nutrition **2**:359, 1930.

30. Rubner, M.: Die Gesetze des Energieverbrauchs bei der Ernährung, Leipzig, F. Deuticke, 1902.

31. Barcroft, J.: Zur Lehre vom Blutgaswechsel in den verschiedenen Organen, Ergebn. d. Physiol. **7**:744, 1908.

32. Zuntz, N.: Verdauungsarbeit und spezifisch-dynamische Wirkung der Nahrungsmittel, Med. Klin. **6**:351, 1910.

33. Verzár, F.: Die Wirkung intravenöser Kochsalzinfusionen auf den respiratorischen Gaswechsel, Biochem. Ztschr. **34**:41, 1911.

34. Tangl, F.: Colorimetrie der Nierenarbeit, Biochem. Ztschr. **53**:36, 1913.

35. Janssen, F., and Rein, H.: Ueber die Zirkulation und Wärmebildung in der Niere, Klin. Wchnschr. **6**:1827 (Sept. 17) 1927.

36. Lublin, A.: Ueber den Einfluss der Nierenarbeit auf den Gaswechsel des Menschen, Ztschr. f. klin. Med. **109**:371, 1929.

37. Grollman, A.: Physiological Variations in Cardiac Output of Man: Changes in Cardiac Output, Metabolism, Blood Pressure, and Pulse Rate of Man Following the Ingestion of Fluids, Am. J. Physiol. **89**:157, 1929.

38. Benedict, F. G., and Carpenter, T. M.: Food Ingestion and Energy Transformations with Special Reference to the Stimulating Effect of Nutrients, Publication 261, Carnegie Institution of Washington, 1918, p. 355.

METABOLIC RESPONSE TO INJURY

Before further consideration is given to the effects of the administration of fluid in cases of shock, a brief review of the alterations in metabolism in cases of secondary shock might be presented. Secondary or traumatic shock is accompanied by a fall in the rate of oxygen consumption (Aub [39] and Henderson and co-workers [40]). In this investigation diminutions of from 30 to 50 per cent of the original rate were commonly noticed. It was found that the extent of the fall of the metabolism seemed to be dependent on the mode of production of shock (Davis [14]). Shock resulting from a single large hemorrhage is charac-

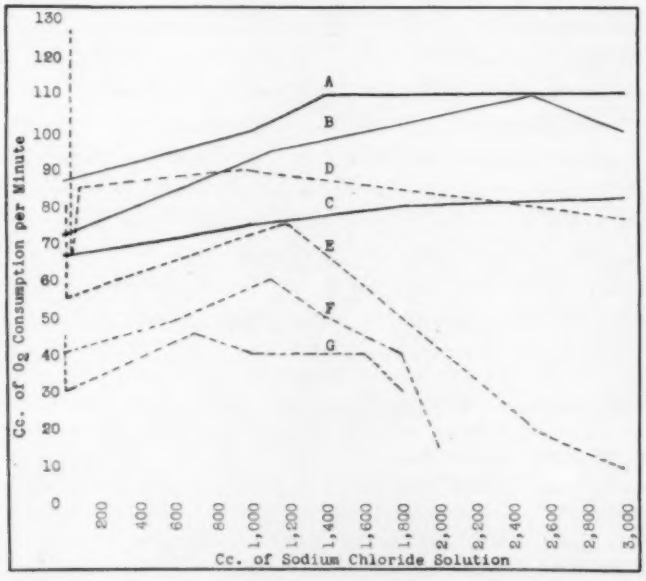

Fig. 1.—Curves showing the influence of the administration of water on the rate of oxygen consumption in normal animals and in animals in shock. *A*, *B* and *C* are the curves for the normal control animals; *D*, the curve for an animal in which shock was produced by extensive hemorrhage; *E*, the curve for an animal in which shock was produced by trauma plus hemorrhage, and *F*, the curve for an animal in which shock was produced by obstruction of the portal vein.

terized by a tendency to rapid and spontaneous recovery. When severe trauma is involved, the metabolic rate has a tendency to remain fixed at a low level, and the return to normal is considerably delayed. Immediately after ligation of the portal vein, the rate of oxygen consumption

39. Aub, J. C.: Studies in Experimental Traumatic Shock: I. Basal Metabolism, Am. J. Physiol. **54**:388, 1920.

40. Henderson, Y.; Prince, A. L., and Haggard, H. W.: Observations on Surgical Shock, J. A. M. A. **69**:965 (Sept. 22) 1917.

is unaffected, sometimes for an hour or more. Thereafter, a progressive downward tendency may be noted, with death as the end-result. The influence of histamine dihydrochloride is variable. Usually a fall in metabolism occurs, which in the experiments reported here has not been great. The decrease in metabolism, however, tends to be maintained for a long period. While these are the primary metabolic phenomena associated with shock, certain later changes must be taken into consideration. Within twenty-four hours an increased nitrogenous output in the urine occurs, and this is associated with a rise in metabolism (Cuthbertson [41]), which continues for periods varying from seven to ten

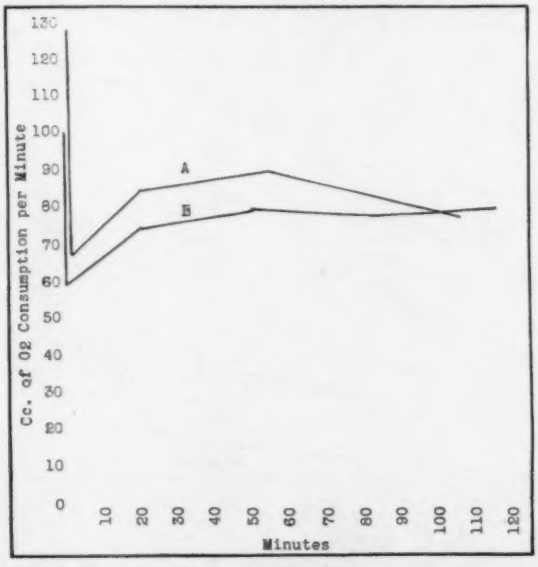

Fig. 2.—Curves showing the metabolism readings of animals which recovered spontaneously from hemorrhage. *A* is the curve of an animal that received saline solution, and *B*, the curve of an animal that did not receive saline solution.

days. This excretion of sulfur creatinine and potassium has been explained by the autolysis of injured tissues at the site of trauma.

ANOXEMIC EFFECTS OF FLUID IN SHOCK

It has been stated already that the metabolic rate of normal animals is increased by the intravenous injection of fluid. The maximal increase varies between 25 and 50 per cent and is reached only after from 1,400 to 2,400 cc. of fluid has been given. Thereafter, a plateau is obtained in the curve, and this is maintained so long as the fluid is

41. Cuthbertson, D. P.: Observations on Disturbance of Metabolism Produced by Injury to Limbs, Quart. J. Med. **1**:233, 1932.

injected and for some time afterward. Generally, the absolute increase in the rate of oxygen consumption varies directly with the initial rate of metabolism. In a normal healthy animal the tolerance for water is great. Fluid to the extent of 25 per cent of the total body weight of the animal may be given without any deleterious result.

In traumatic shock a peculiar alteration of the metabolic response to fluids takes place. The maximal increase in the rate of oxygen usage is reached at an earlier stage during the injection of fluid, i. e., after from 700 to 1,200 cc. of the solution has been given. Thereafter, if more fluid is given, a progressive decline in the rate of oxygen consumption takes place. Apparently in shock tolerance for fluids is much diminished, as the administration of a quantity of water equivalent to only from

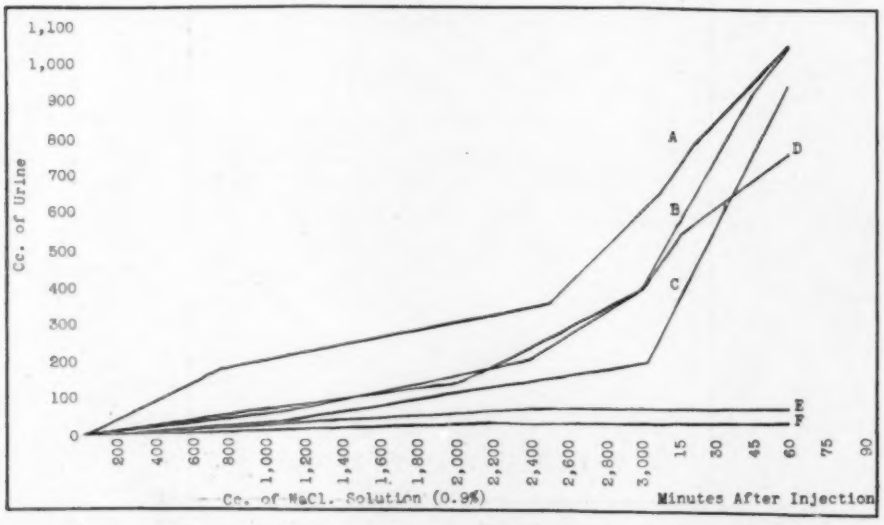

Fig. 3.—Curves showing the urinary secretion of animals in shock in relation to the administration of fluids. *A*, *B* and *C* are the curves of normal control animals. *D* is the curve of an animal in which shock was produced by the injection of histamine dihydrochloride; *E*, the curve of an animal in which shock was produced by hemorrhage, and *F*, the curve of an animal in which shock was produced by trauma.

5 to 12 per cent of the body weight produces a fall in the metabolic rate. In this investigation it was noted that the degree of intolerance for fluids varied with the depth of shock induced. It was particularly noticeable in those animals exposed to severe trauma. Hemorrhage in small amounts did not alter the metabolic response, as such animals reacted in almost a normal manner. Shock produced by ligation of the portal vein invariably diminished the tolerance of the animal for fluid.

The reason for the diminished tolerance for water of the animal in shock is difficult to state. Certainly, previous investigations point to

the fact that the stimulant action of fluids on the metabolism is greatest while the fluid is in the blood vessels, and therefore can be attributed to an increase in the blood volume, rate of blood flow or both (Davis [42]). In shock, the fluid leaves the vascular bed rapidly, in spite of the existing concentration of the blood. This explains the ephemeral nature of the stimulant action of water on the rate of oxygen consumption, but it does not explain the depressant action of water in traumatic shock.

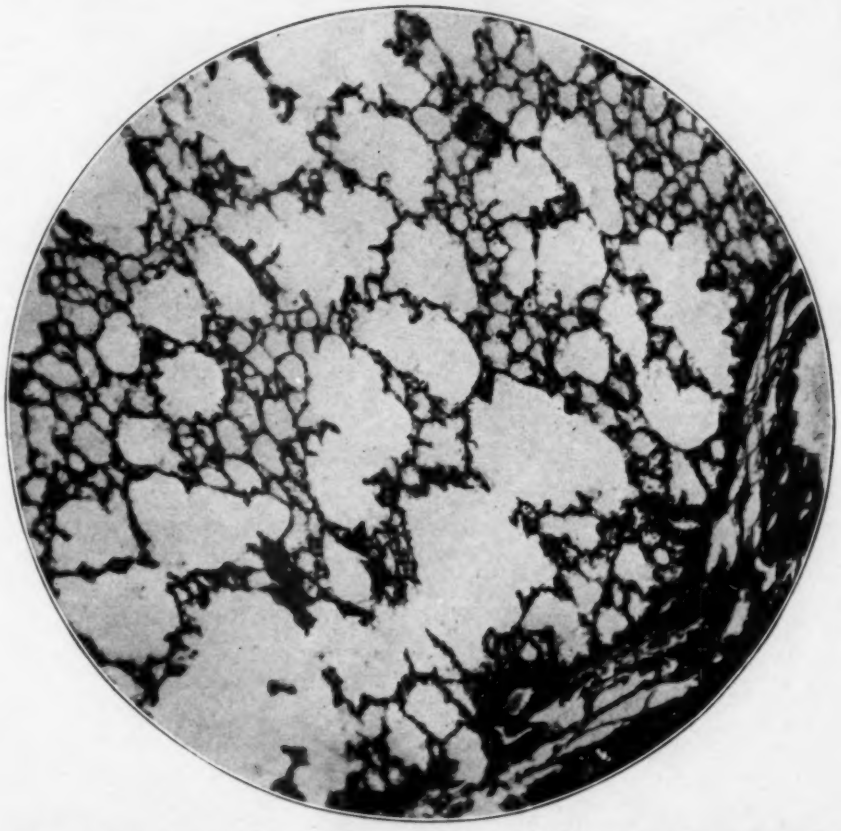

Fig. 4.—Photomicrograph of a section of the lung of an animal in which shock was produced by trauma; × 50. Acute congestion of the alveolar capillaries and minimal alveolar edema are evident.

Anoxemia, which is already present in shock, is intensified with amounts of fluid which would not affect a normal animal. In some way the transport, transfer or utilization of oxygen is affected.

42. Davis, H. A.: Studies in Water Balance: I. Excessive Oxygen Usage Response of Dehydrated Animals to Water and Electrolytes, Proc. Soc. Exper. Biol. & Med. **33**:242, 1935.

RENAL FUNCTION IN SHOCK

In secondary shock not only is there an alteration in the metabolic response, but there is also a change in the renal function. The injection of a 0.9 per cent solution of sodium chloride into a normal animal was followed by a brisk diuresis, which commenced shortly after the initiation of the injection. Within one hour after completion of the infusion,

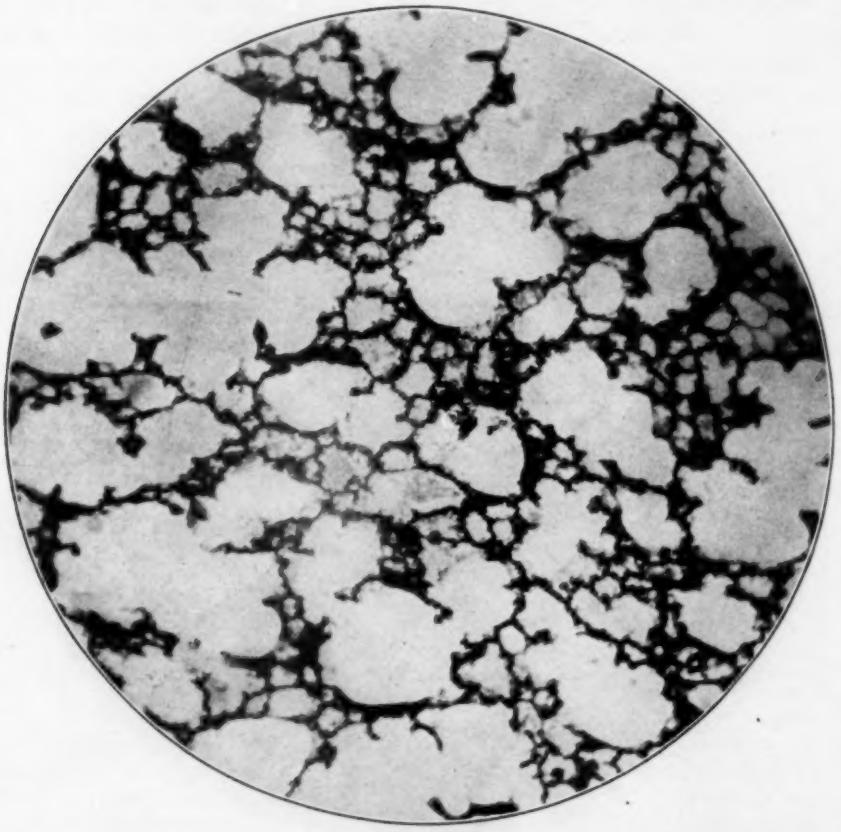

Fig. 5.—Photomicrograph of a section of a lung of an animal with traumatic shock which was treated by the administration of fluids; × 50. Acute congestion of the alveolar capillaries and moderate alveolar edema are evident.

the urinary secretion was abundant, and volumes equivalent to one third or one half of the total amount of fluid given were obtained. In secondary shock, water diuresis is minimal, even after the infusion of large amounts of fluid. The extent of the water diuresis varies inversely with the depth of shock present.

While acidosis favors the release of water from tissues (Davis and Dragstedt [43]), its presence in association with traumatic shock is of little value. This traumatic anuria is probably related to the lowering of the filtration pressure within the glomerular capillaries. Histologic studies of kidneys removed from animals in which shock had been produced reveal the presence of an acute cloudy swelling of the tubular

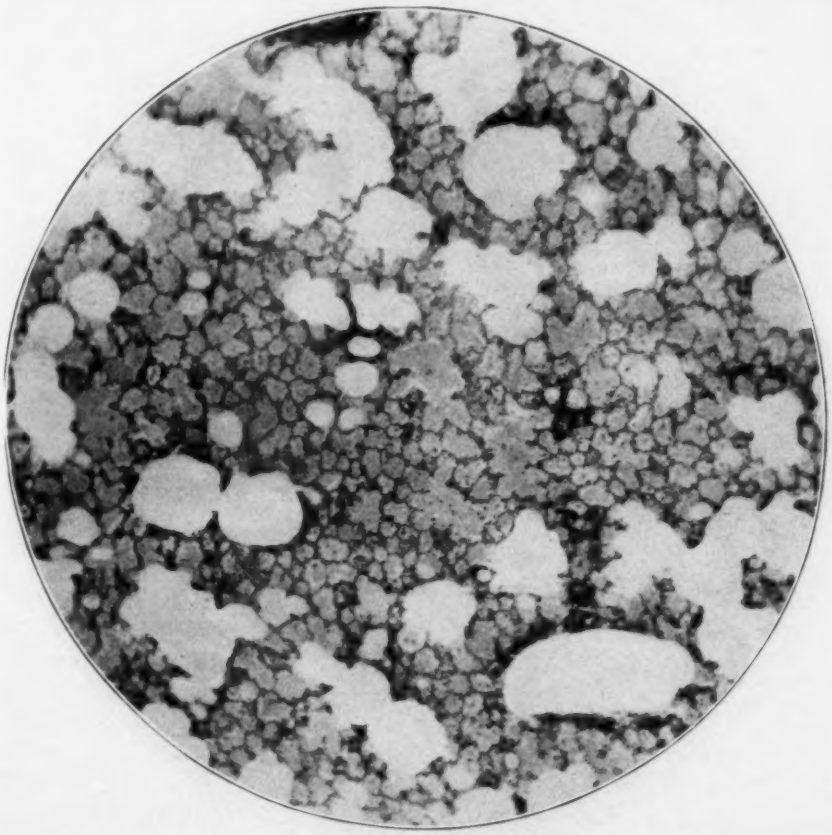

Fig. 6.—Photomicrograph of a section of the lung of a normal animal treated with fluids; × 50. No congestion of the alveolar capillaries is evident, but extensive alveolar edema may be seen.

epithelium with pyknosis of nuclei. The glomerular capillaries are greatly congested, but no specific lesions are demonstrable. These alterations in the tissues are reversible, and no permanent renal injury

43. Davis, H. A., and Dragstedt, L. R.: The Relative Significance of Electrolyte Concentration and Tissue Reaction in Water Metabolism. Am. J. Physiol. **113**:193, 1935.

occurs. However, the presence of anuria is significant, as it emphasizes the inability of the organism in shock to distribute freely within itself ingested fluids.

NATURE OF THE ANOXEMIC RESPONSE

The exact mechanism of production of the anoxemia resulting from the administration of fluids in cases of secondary shock is debatable.

Fig. 7.—Photomicrograph of a section of the liver of a normal animal treated with fluids; × 50. Intense edema of the liver cells, with compression of the liver capillaries, and clear nuclei without pyknosis are evident.

A diminution of oxygen usage might result from (1) an insufficiency of oxygen available to the blood in its passage through the lungs, (2) a decrease of available oxygen-carrying material and (3) interference with oxygen utilization by the cells. The third cause may, with justification, be excluded in view of the fact that the administration of large quantities of fluids sufficient to produce obvious generalized edema of

the tissues in normal animals causes no decrease in the rate of oxygen consumption. It is possible, however, that in shock the utilization of oxygen by anoxemic tissues may be diminished by the presence of large amounts of inert fluid.

Attention was focused on the first two of these possible causes. Two subsidiary investigations were therefore undertaken. In order to examine the factor of pulmonary changes in the production of anoxemia

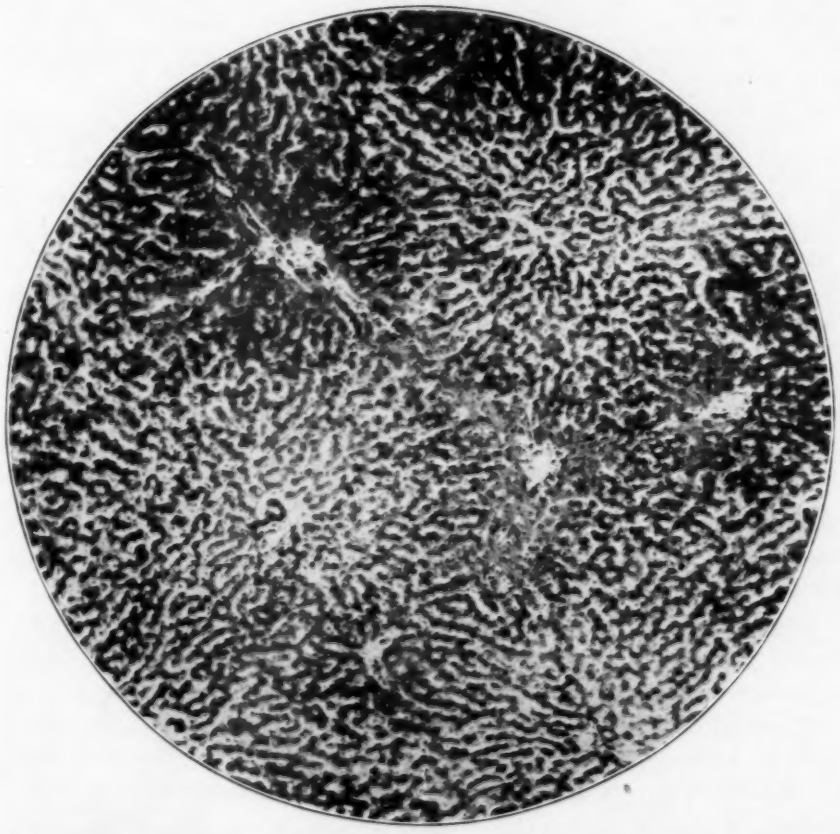

Fig. 8.—Photomicrograph of a section of the liver of an animal with traumatic shock which was treated by the administration of fluids; × 50. Acute congestion of the central capillaries with compression of the liver cells and slight nuclear pyknosis at the periphery of the nodules are evident.

resulting from shock with and without the administration of fluids, the following experiment was designed:

A series of healthy dogs weighing from 8 to 20 Kg. was used. Pentobarbital sodium was used in a dosage of 0.03 Gm. per kilogram of body weight. Shock was induced (1) by repeated trauma to both hindlimbs and (2) by repeated

hemorrhage. Kymographic tracings of the blood pressure were made. When the blood pressure was lowered to 60 mm. of mercury or less, the animal was considered to be in shock. Usually the blood pressure was maintained in the neighborhood of from 40 to 50 mm. of mercury. In order to obviate any pulmonary changes due to prolonged immobilization, the period of shock was not permitted to exceed one and one-half to two and one-half hours. Each animal was moved into various positions during the experiments. Each animal was killed either by intensification of the shock present or by air embolism, and the lungs and other viscera were immediately removed and placed in a hot diluted solution of formaldehyde U. S. P. (1:10). Four groups of experimental animals were studied: group a, animals given pentobarbital; group b, normal animals that received fluid; group c, animals in shock, and group d, animals in shock that were given fluids.

Histologic changes were absent in the lungs of the animals of group a. In the animals in group b edema was present in the alveolar spaces. This was not uniform in distribution, however, and little or no cellular reaction was observed. The histopathologic picture found in the animals of group c will be described in a forthcoming paper. The findings pertinent to the discussion here will be described. In animals in shock edema was present in scattered areas throughout both lungs. But it was not great in amount and was less than that found in the lungs of normal animals receiving fluids. In the animals of group d edema was not conspicuous and was less than that present in the animals of group b.

These findings provide no basis for the assumption that the anoxemia of shock results from pulmonary edema. Moreover, edema of the lungs does not seem to be a significant factor in the production of the anoxemia following the injection of fluids into the animal in shock.

Changes in the tissues resulting from the administration of fluids depend primarily on the state of the vascular bed. When fluids enter the circulatory system in amounts sufficient to increase the volume of circulating blood, adaptation mechanisms are at once brought into play. In table 1 are described briefly certain visceral changes accompanying the administration of fluid to normal animals and to animals in shock.

This brief summary of the visceral changes in animals in shock which received fluid and in those which did not receive fluids reveals a paradoxical phenomenon. In the animals with traumatic shock, a condition in which capillary permeability is increased, the administration of fluid produced less pulmonary edema than in the normal animals. This paradox is readily explainable by the fact that in shock much of the fluid escapes from the circulatory system at the point of least resistance—the area of trauma. It was noted in these animals that the traumatized limbs became very edematous, which would be consistent with the finding of only mild pulmonary edema, relatively little peri-

toneal transudate and the histologic evidence of water storage localized to the central portion of the hepatic lobules.

The final part of this investigation was undertaken to determine to what extent the use of fluid could produce a loss of protein and cellular elements from the blood of normal animals and of animals in shock. Standard amounts of fluid (60 cc. per pound of body weight) were given to the animals by vein, and the peritoneal transudate was

TABLE 1.—*Tissue Changes Resulting From Administration of Fluid in Normal and Traumatized Animals*

Viscera	Normal Animal; Fluid Administered	Animal in Shock	Animal in Shock; Fluid Administered
Lung	Considerable general edema fluid in alveoli; no capillary congestion; no cellular reaction in alveoli; no petechial hemorrhage	Mild discrete patchy distribution of edema in alveoli; marked cellular reaction with mononuclear and polymorphonuclear cells; acute congestion of alveolar capillaries with diapedesis of red blood cells; areas of hemorrhage and petechiae	Mild edema of alveoli with considerable cellular reaction—monocytes and polymorphonuclears; acute congestion of alevolar capillaries with diapedesis of red blood cells; areas of hemorrhage in alveoli; subpleural petechiae
Kidney	Slight increase in size; distention of Bowman's capsule; no glomerular lesions; tubular epithelium normal with large vesicular nuclei and well defined chromatin material	Slight diminution in size; acute cloudy swelling of tubular epithelium with small pyknotic nuclei; congestion of glomerular capillaries	Early cloudy degeneration of tubular epithelium with small pyknotic nuclei; no glomerular lesions
Liver	Larger than normal; marked edema and vacuolization of liver cells; nuclei large, clear and vesicular, with well defined chromatin material; moderate capillary dilatation in region of central vein	Slightly larger; marked congestion of liver capillaries in region of central vein; liver cells small, compressed with small pyknotic nuclei	Slightly larger; mild edema and vacuolization of hepatic cells around central vein where nuclei are large and vesicular; cells stain deeply with small pyknotic nuclei in periphery of lobule
Spleen	Enlarged; edema of splenic pulp spaces; dilatation of splenic sinusoids	Small; congestion of splenic sinusoids	Small; congestion of splenic sinusoids
Heart	Moderate dilatation—right auricle and ventricle; no histopathologic changes	Contracted; no histopathologic changes	Moderate dilatation; no histopathologic changes
Pancreas	No specific pathologic process	No specific pathologic process	No specific pathologic process

carefully measured and examined for total proteins, albumin and globulin and for erythrocytes after centrifugation.

The representative data, shown in table 2, suggest that in the intact animal the administration of fluid leads to a loss of albumin and globulin from the blood stream. The loss of protein into the peritoneal fluid is 9.5 per cent (calculated on the basis of a blood serum content of 7 Gm. per hundred cubic centimeters of blood). The percentage of protein lost in the animal in shock that is treated with fluid is equal to, and probably greater than, that found in the intact animal. It will be

noted that in dogs 3 and 4 the percentage calculations have been made on the basis of normal calculated volumes of blood. As the volume of blood is reduced in traumatic shock, the percentage of protein lost in animal 3 is probably higher than is shown by table 2.

COMMENT

This study seems to indicate the presence of certain physiologic and pathologic limitations to the free and equal distribution of fluids in the organism in shock. The introduction of fluid into the intact animal produces a dilution of the blood with subsequent diuresis. With larger amounts of fluid, the liver cells imbibe water from the blood stream and become edematous, ultimately causing compression of the hepatic capillaries. Still later, the splenic sinusoids dilate. A transudate of protein-laden fluid occurs into the peritoneal cavity. Finally, the fluid containing serum albumin and globulin permeates through the capillary walls, particularly those of the lungs, leading to pulmonary edema. In secondary shock the normal reservoir action of the blood for fluids is lost, and fluid leaves the blood stream more rapidly than in the intact animal (Davis [44]). The site of fluid loss is greatest in the region of trauma, so that relatively little transudation of fluid occurs into the peritoneum or into the alveoli of the lungs. Evidences of water imbibition are present in the central portions of the hepatic lobules. It cannot be stated definitely here whether or not this localization of water storage by the liver is indicative of inability of the liver cells to store water or of an insufficient quantity of water available for storage. The production of a lowered rate of oxygen consumption by the administration of fluids in cases of secondary shock does not result from interference with the supply of available oxygen by pulmonary edema. The stimulant action of water on metabolism is exerted only so long as fluid is actually present

TABLE 2.—*Effect of Sodium Chloride Solution (0.9 Per Cent) on Protein Loss into Peritoneal Cavity*

Dog No.	Type of Experiment	Calculated Blood Volume, Cc.	Calculated Total Protein in Blood, Gm.	Peritoneal Fluid, Cc.	Peritoneal Fluid			
					Total Protein, Gm. per 100 Cc.	Albumin, Gm. per 100 Cc.	Globulin Gm. per 100 Cc.	Protein Loss, %
2	Control animal; fluid administered	738	51.0	250	4.87	3.78	1.09	9.5
3	Animal in shock; fluid administered	553	38.5	100	3.50	2.70	0.80	9.3
4	Animal in shock (traumatic)	1,353	94.5	10	5.70	4.20	1.50	0.6

44. Davis, H. A.: The Behaviour of Isotonic and Hypertonic Solutions in the Blood Stream of Normal and Dehydrated Animals, Proc. Soc. Exper. Biol. & Med. **32**:210, 1934.

within the circulatory system and seems to depend on the resultant increase of blood volume or blood flow (Davis [42]). Removal of serum albumin and globulin and of blood cells by the introduced fluid into the area of trauma and peritoneal cavity diminishes the material available for transportation of oxygen and leads to a still greater degree of oxygen want.

CONCLUSION

In traumatic shock a peculiar alteration of the response to fluids exists. The physiologic availability of water is diminished even to the point of actual intolerance (Davis [45]). Even the transfusion of blood, as has been pointed out by Zunz and Govaerts,[46] can be harmful. The administration of a quantity of fluid equivalent to from 5 to 12 per cent of the body weight may result in a fall in the metabolic rate. The intact animal, on the contrary, may be given fluid to the extent of 25 per cent of the total body weight without any diminution in the rate of oxygen consumption. The anoxemia of traumatic shock is augmented by the administration of fluids, which promote a loss of protein and cells into the traumatized area, into the peritoneal cavity and, to a slight extent, into the alveoli of the lungs and general tissue spaces.

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45. Davis, H. A.: Influence of Water Administration upon Oxygen Consumption Rate in Shock, Proc. Soc. Exper. Biol. & Med. **34**:21, 1936.

46. Zunz, E., and Govaerts, P.: Recherches expérimentales sur les effets de la transfusion dans les divers états de collapsus circulatoire, Bull. Acad. roy. de méd. de Belgique **29**:796, 1919.

EXTENSIVE BURNS

TREATMENT WITH SILVER NITRATE AND METHYL ROSANILINE

HIRA E. BRANCH, M.D.
DETROIT

The ideal treatment for burns is one that, first, saves the patient's life; second, eliminates pain, shock and toxemia; third, reduces the morbidity to a minimum, and fourth, allows the burned area to heal quickly without skin grafting being necessary.

The method of treating burns to be brought out in this paper approaches closely to the "ideal" treatment, as may be seen by the accompanying table. First, there have been no deaths in cases in which the treatment has been used thus far, and there were only seven deaths in ninety-five cases in which this form of therapy was used during its development. Second, the period of pain and shock was very short. Toxemia did not develop as a serious threat. Third, the morbidity was greatly reduced, for the patients were turned about in a few hours and were urged to get up and about in a few days and to wait on and amuse themselves. Fourth, skin grafting was done in only one case. However, the mere use of this combination treatment for the local area did not do all the aforementioned things alone; naturally measures for the general upbuilding of the patient were also carried out.

BRIEF RÉSUMÉ OF MODERN TREATMENTS FOR BURNS

In 1925 the late Dr. Edward C. Davidson[1] published his famous paper entitled "Tannic Acid in the Treatment of Burns," in which the use of compresses on the injured areas was advised, a 2.5 per cent aqueous solution of tannic acid being employed. Attempts to make a satisfactory ointment of tannic acid at that time failed, chiefly because an oily base was used. Later, a spray of a 5 per cent aqueous solution of tannic acid was used in place of the compresses. There is now little question but that the spray did revolutionize the manner of dealing with burns. The mortality rate has been lowered from 35 or 38 per cent to 10.5 or 12 per cent. Later, this treatment was altered somewhat by the use of a 5 per cent tannic acid gel, a mixture of tannic acid in a water-

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The experimental work for this monograph was done at the Children's Hospital of Michigan, while the author was in the surgical service.

1. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg., Gynec. & Obst. **41**:202-221 (Aug.) 1925.

soluble base. The principles of treatment are the same with the gel as with the solution, but the tanning process is much slower with the former.

There were little or no changes of note in the management of burns from 1925 till 1932, as far as the treatment of the local area was concerned. Big strides were made in treating the general condition of the patient, the water balance and the chloride and plasma protein contents being carefully watched. However, this has been dealt with elsewhere. In 1932 Dr. Robert Henry Aldrich [2] read a paper in Boston on "The Role of Infection in Burns," which dealt with the theory and treatment of burns with special reference to methyl rosaniline (gentian violet). The mortality rate for burns treated with this drug dropped to the same level as that for burns treated with tannic acid, namely, from 10 to 12 per cent. The methyl rosaniline was sprayed on the burned area in the form of a 1 per cent aqueous solution. Again a gel was made, the methyl rosaniline being used in a water-soluble base. This gel has been used chiefly in the treatment of ambulatory patients with minor burns and is an excellent way of dealing with them. A generous coat of the gel is spread over the burn, and a bandage is applied which is not removed for about ten days. Practically all burns are cleanly healed in that time.

The next step forward was taken by Dr. A. G. Bettman [3] in 1935, when he combined the use of a solution of silver nitrate over one spray of tannic acid, thus obtaining an immediate black crust over the injured surface. This saved about four hours in the tanning process. However, the disadvantages and the advantages of this method were the same after the first few hours as those of the tannic acid alone. Perhaps it is now advisable to list the advantages and disadvantages of the aforementioned treatments.

ADVANTAGES AND DISADVANTAGES OF VARIOUS METHODS OF TREATMENT

Tannic Acid.—Herewith are quoted some of Dr. Davidson's conclusions as to the advantages of treatment with tannic acid:

> Coagulation of the devitalized tissue by the use of tannic acid lessens toxemia.
>
> Tannic acid as an initial dressing has an analgesic effect.
>
> The subsequent use of the open air method causes minimal trauma and promotes general comfort.

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2. Aldrich, R. H.: The Rôle of Infection in Burns, New England J. Med. **208**:299-309 (Feb. 9) 1933.

3. Bettman, A. G.: Tannic Acid-Silver Nitrate Treatment of Burns, Northwest Med. **34**:46-51 (Feb.) 1935.

4. Davidson, E. C.: The Prevention of the Toxemia of Burns: Treatment by Tannic Acid Solution, Am. J. Surg. **40**:114-115 (May) 1926. Davidson.[1]

The local astringent effect prevents the loss of body fluid.

Secondary infection is limited by this method of treatment because of the absence of a favorable nidus for bacterial growth.

Scar tissue formation has been lessened.

The protective layer of coagulated protein forms a scaffold for the growth of the young epithelial cells over the denuded surface.

The tannic acid treatment has brought a decided drop in the mortality rate for burns.

A uniform method of dealing with burns has been established throughout the world.

Some of the disadvantages of the treatment with tannic acid are:

1. The patient must remain quiet while covered with the black coagulum, otherwise cracking occurs. This stationary position favors pneumonia and aural infections and does not promote the "will to live" spirit so often needed by the patient with an extensive burn.

2. Cracking of the coagulum brings about infection, which usually requires removal of the crust.

3. The coagulum sticks tenaciously to the third degree areas of the burn, requiring surgical removal. (The soaking of a tannic acid crust is contraindicated because of infection and toxemia.)

4. Surgical removal of the black coagulum destroys many islands of epithelium beneath, which invariably necessitates skin grafting to the denuded areas.

Methyl Rosaniline Spray.—The advantages of using a spray of methyl rosaniline solution are the same as for tannic acid, with the following differences:

1. The crust that forms is pliable, but if it should crack the area is merely resprayed and infection is limited.

2. Infection is prevented, and if it is present before treatment is started methyl rosaniline helps to eradicate it.

3. Débridement of the burn does not have to be as severe.

4. The crust formed by methyl rosaniline can be soaked off, thus preserving the epithelial islands beneath. This practically eliminates skin grafting.

The disadvantages of this type of treatment are:

1. The solution has no astringent effect and does not prevent loss of body fluids.

2. The crust takes from one to three days to form, owing to the fact that the devitalized proteins are not coagulated.

Silver Nitrate-Tannic Acid Treatment.—Except for the rapid tanning effect and the saving of a few hours in that respect, the advantages and the disadvantages of this combination of drugs are practically the same as those for tannic acid alone.

EVOLUTION OF THE SILVER NITRATE-METHYL ROSANILINE TREATMENT

The principles of the treatment of burns with tannic acid are sound. This has been proved by the late Dr. Davidson and others. Clear evidence has been shown by the decrease in mortality from about 35 per cent to about 10 or 12 per cent. However, the solution is contraindicated in infected areas or surfaces that bend or are subject to infection, such as the perineum, fingers, toes, scalp, etc. During 1935 I experimented with various combinations of treatments, always using tannic acid as the basic treatment, chiefly because of its beneficial qualities and also because Dr. Grover C. Penberthy, with the late Dr. Davidson, had thoroughly established that form of treatment at the Children's Hospital of Michigan, in Detroit, where this work was done.

The first combination tried was the use of a spray of a 5 per cent solution of tannic acid on all clean areas that did not require motion or were not subject to infection. A 1 per cent aqueous solution of methyl rosaniline was used on all areas that were either infected or likely to become infected. The results were a great improvement over those obtained with tannic acid alone. The next modification was the combination of silver nitrate with the tannic acid on the clean areas and methyl rosaniline on the potentially dirty surfaces. The results were about the same as with the first combination.

The disadvantages have all been listed previously. However, the main ones were that the patient had to remain quiet for from two to three weeks, sometimes with his extremities in slings off the bed. The crust formed by the tannic acid had to be removed surgically from the deeper burned areas, necessitating skin grafting. The methyl rosaniline did not form an immediate crust and stop the loss of body fluids. The next problems were to find a combination which not only would stop these abuses but would keep the mortality rate down, prevent pain, shock and toxemia, reduce the morbidity (absolute rest in bed chiefly), and lastly, allow the burned surfaces to heal quickly without skin grafting. The methyl rosaniline prevented infection and toxemia and reduced morbidity and the amount of skin grafting, but the pain, shock and loss of body fluids were not eliminated, and the mortality rate was still about 10 to 12 per cent. The crusts formed by the tannic acid prevented pain and toxemia if infection did not occur. However, the

morbidity and skin grafting were not lessened, and the mortality rate remained about the same. Methyl rosaniline did not mix with tannic acid to give any advantageous qualities. Silver nitrate with tannic acid merely formed a black crust immediately over the burned surface. This black crust was due partly to a precipitation of the tannic acid as silver tannate and partly to a coagulation of the devitalized proteins.

The next combination used was silver nitrate and methyl rosaniline. The results were excellent. The 10 per cent solution of silver nitrate acted on the proteins of the serum exuding from the burned area to immediately lay down a milky white coagulum. This then could take the place of tannic acid in coagulating the proteins and preventing the loss of body fluids. The methyl rosaniline stained this coagulum violet and aided in destroying and preventing infection. It was found at the end of three weeks that the second degree burns had all healed, and the crust had cracked off. The third degree areas were still covered by the violet coagulum, but this was easily removed by bathing daily in brine and by the use of compresses. On soaking off the crust there were invariably enough epithelial islands beneath so that no skin grafting was necessary. After the coat was off compresses soaked in cysteine hydrochloride, 1 : 200 solution, or compresses or gauze treated with scarlet red was used to stimulate epithelization of the denuded areas. Thus the period of pain and shock was practically eliminated. Infection and toxemia rarely developed. The amount of parenterally administered fluids necessary was greatly reduced. The morbidity was markedly decreased, for the patients could be freely turned after a few hours, and after the first few days they could be removed from the burn tent and allowed to walk about. Their general spirits improved greatly.

During 1935, the transition period from treatment with tannic acid to the new silver nitrate-methyl rosaniline method, there were ninety-five patients treated whose burns were severe enough to necessitate hospitalization. Of these, seven died, giving a mortality of about 7.3 per cent. In 1936, through June, eighteen patients with burns severe enough to necessitate hospitalization were treated by the silver nitrate-methyl rosaniline method. The results are presented in detail in the accompanying table. There were no deaths; all the patients were up and about during their recovery, unless they were too young to walk. The period of hospitalization was very short. Only one patient required a skin-grafting operation. The highest temperature was 105.4 F. (rectal), the temperature usually ranging from normal to about 100.4 F. (rectal). Most burns had been covered with various oils, ointments or home remedies before the silver nitrate-methyl rosaniline treatment was employed.

Data on Burns Treated by Silver Nitrate and Methyl Rosaniline

Case	Sex	Color	Age*	Age of Burn, Hr.	Etiology	Previous Treatment	Degree of Burn	Extent of Body Surface, %	Location	Period of Complete Rest in Bed	Period of Hospitalization, Days	Highest Temperature (R.)	Average Temperature (R.)	Skin Grafting	Results
1	M	W	2½	24	Fire	Olive oil	2-3	20	Head; neck; back	5 days	27	102.0	100.0	0	Recovery
2	M	W	4	4	Coffee	Oil	2-3	20	Chest; right thigh; abdomen; perineum	2 days	18	102.0	99.4	0	Recovery
3	M	W	1½	48	Water	Ointment	2	15	Buttocks; back; thighs	8 days	13	101.6	99.0	0	Recovery
4	M	W	8	24	Water	Ointment; oil	3	35	Head; neck; back; arms	6 days	62	102.0	100.0	1	Recovery
5	F	W	3½	48	Fire	None	2	12	Face; chest; left arm	1 day	10	101.6	99.8	0	Recovery
6	M	W	11	9	Coffee	Lard; ointment	2-3	15	Abdomen; perineum; thighs	5 days	18	102.4	100.0	0	Recovery
7	M	W	10 mo.	1	Water	None	3	18	Head; trunk; left arm	8 days	32	105.4	100.8	0	Recovery
8	F	W	9	1	Water	Ointment	2-3	10	Left side of body; arm; leg	1 day	6	101.4	99.8	0	Improvement
9	M	W	1	20	Coffee	Ointment	3	12	Chest; right arm	7 days	12	102.4	99.8	0	Recovery
10	M	W	2½	1	Water	None	2-3	30	Trunk; right arm; left leg	2 days	14	100.4	99.0	0	Improvement
11	M	W	7 mo.	11	Cleaning solution	Tannic acid gel	2-3	25	Trunk; thighs	1 day	22	104.8	100.6	0	Recovery
12	M	N	2	1	Water	None	2-3	25	Neck; trunk; arms	5 days	22	104.4	101.0	0	Recovery
13	F	W	8	2	Fire	None	2	8	Face; neck; chest	4 hours	13	101.2	99.0	0	Recovery
14	M	W	1	1	Water	Oil; ointment	3	35	Head; trunk; right arm	6 days	35	104.0	100.4	0	Recovery
15	M	N	7	1	Water	Oil	2	8	Head; neck; chest	2 days	16	102.6	99.4	0	Recovery
16	F	W	2	1	Water	Oil; tannic acid ointment	2-3	12	Back; perineum	1 day	18	100.2	99.0	0	Recovery
17	F	W	1	3	Water	Ointment	2	10	Arms; legs; chest	2 days	6	102.2	100 0	0	Improvement
18	M	W	1	2	Water	None	2	5	Left leg; perineum	3 days	8	100.6	99.6	0	Improvement

* Unless otherwise stated, the age is given in years.

ROUTINE TREATMENT WITH SILVER NITRATE-METHYL ROSANILINE

After the patient is burned, the following procedures are carried out:

1. A large dose of sedative is given—codeine or morphine sulfate with phenobarbital.

2. The burned areas are washed with tincture of soft soap.

3. Débridement of the burn is done lightly and rapidly by merely breaking the blisters and removing the loose skin. The area is then washed with a sterile saline solution or a solution of boric acid.

4. The burn is sprayed at once with a 1 per cent aqueous solution of methyl rosaniline, and after three or four minutes the entire burned area is swabbed with a 10 per cent solution of silver nitrate.

5. The patient is placed in a burn tent at a temperature of 85 to 90 C. The area is resprayed with a 1 per cent solution of methyl rosaniline every fifteen minutes for about five times. After that the spray is used once or twice a day only if necessary.

6. Fluids are forced strongly by mouth.

7. Hypodermoclysis of physiologic solution of sodium chloride is given every six or eight hours, as indicated by the patient's condition.

8. Fluids are given intravenously only if the temperature rises to 103 F.

9. Transfusion is done if necessary.

10. The patient is moved about as soon as the crust is established and is allowed to walk about as soon as the period of shock is over and if no signs of toxemia develop. The patient is gotten up and about regardless of the size or location of the burned area and usually may walk about freely after the first twenty-four hours if the burn is small, covering up to about 20 per cent of the body surface. It is perhaps safer to keep the patient with the more extensive burn in bed from five to seven days, or at least till the temperature has been normal for about twenty-four hours.

11. If any coagulum remains at the end of two and one-half or three weeks, it should be soaked off with brine or by compresses. Compresses treated with cysteine hydrochloride, 1:200 solution, may then be applied to the third degree areas that are not healed, alternating every four days with compresses soaked in boric acid or gauze treated with scarlet red may be applied to stimulate epithelization.

SUMMARY AND CONCLUSIONS

A treatment for extensive burns which employs silver nitrate and methyl rosaniline has been presented in detail.

During the experimental period ninety-five patients were treated with various combinations of solutions, and the mortality rate was only 7.3 per cent.

Since the establishment of the silver nitrate-methyl rosaniline therapy, eighteen additional patients have been treated, with no deaths. The data in these cases have been presented in detail.

A simple method of dealing with minor burns in ambulatory patients has been presented, methyl rosaniline being used in a water soluble base.

The principles set up for the treatment of burns with tannic acid have been upheld with this form of therapy, even though that solution was not used, and, in addition, there are several advantages.

Shock and pain were held at a minimum.

Toxemia rarely developed as a clinical threat.

Infection rarely became a severe complication, regardless of the location of the burned area.

Patients were moved about freely after the first few hours and were urged to get up in wheel chairs or to walk in a few days regardless of location of the coagulum or its extent.

Burns healed quickly; the period of hospitalization was very short.

Skin grafting was rarely, if ever, necessary to get the patient quickly covered with epithelium.

Scar tissue was held at a minimum.

Urinalyses done as a routine showed no renal complications.

The general outlook of the patients was extremely good as soon as the period of pain and fright was over.

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MENINGOCOCCIC MENINGITIS COMPLICATING FRACTURE OF THE SKULL

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Acute purulent meningitis complicating fracture of the skull and caused by pyogenic cocci is well known, but meningococcic meningitis occurring under similar conditions has been infrequently reported. Four such cases have recently been observed at the Bellevue Hospital and are being reported because of their therapeutic indications and forensic importance.

A study of the relationship of trauma and fracture of the skull to meningococcic meningitis was reported by Seligmann and Pieper.[1] Of 674 cases of meningococcic meningitis, a history of trauma was recorded in 55, but fracture of the skull was demonstrated in only 2. In 21 of their cases, including the 2 in which there was fracture of the skull, meningitis appeared from one to three days after trauma. In 17 cases trauma preceded the onset of meningitis by from eight days to three months, and in 17 others a period of from four months to two years elapsed between trauma and the onset of meningitis. The authors asserted that in several of these cases the causal relationship between trauma to the head and meningococcic meningitis appeared unequivocal and that in the majority of the others the existence of this relationship was at least probable.

In 8 other cases a similarly long interval (from twelve days to eight months) is recorded between the time of trauma to the head [2] or frac-

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1. Seligmann, E., and Pieper, E.: Die Cerebrospinalmeningitis in Preussen in den Jahren 1923 und 1924, Veröffentl. a. d. Geb. d. Med.-Verwalt. **20**:441, 1926.

2. Reiche, F.: Der senile Typus der übertragbaren Genickstarre, München. med. Wchnschr. **56**:1829 (Sept. 7) 1909. Gutzeit, K., and Stern, R.: Trauma und epidemische Meningitis, Med. Klin. **25**:1400 (Sept. 6) 1929.

ture of the base of the skull [3] and the onset of meningococcic meningitis. In other reports, however, the interval was briefer. In 7 cases [4] meningococcic meningitis followed fracture of the skull within from one to eight days, and in 5 cases of trauma to the head but no fracture of the skull [5] from one to four days elapsed before the development of meningococcic meningitis. In a case of trauma to the head with fracture of the nasal bones [6] meningococcic meningitis was observed eighteen hours after the injury.

REPORT OF CASES

CASE 1.—M. Z., a 52 year old white man, was admitted to the psychiatric service on April 16, 1934, because of an injury to the head. He appeared acutely alcoholic and showed contusions of the nose, epistaxis and projectile vomiting. The temperature was normal, and the spinal fluid, bloody. On April 18 the temperature rose to 103 F., but the spinal fluid was unchanged. On April 19 the spinal fluid was xanthochromic. On April 21 there were stiffness of the neck and a positive Kernig sign, and the spinal tap revealed purulent fluid containing micro-organisms which were identified on culture as meningococci.[7] Three addi-

3. Schmidt, P.: Pneumokokken- und Meningokokken-Meningitis nach Schädelbasisfraktur, Deutsche med. Wchnschr. **42**:124 (Feb. 3) 1916. Terbrüggen, R.: Ein weiterer Fall von epidemischer Zerebrospinalmeningitis nach Schädelbasisfraktur, Ztschr. f. Laryng., Rhin., Otol. **18**:312 (Aug.) 1929. Gruber, G. B.: Zur Lehre von Wesen, Verbreitung und Bekämpfung der Meningokokken-Meningitis, Ztschr. f. Hyg. u. Infektionskr. **80**:219 (Aug. 18) 1915. Lode, A., and Schmuttermayer, F.: Traumatische Meningokokken-Meningitis, Wien. klin. Wchnschr. **42**:5 (Jan. 4) 1929. Macgraith, B. G., and Adel, M. B.: Meningococcal Meningitis Following Fracture of the Skull, Lancet **1**:863 (April 13) 1935. Aronowitsch, G. D.: Meningitis und Trauma (Ueber Fälle von Meningokokken- und Pneumokokkenmeningitis nach Kopfverletzung), Deutsche Ztschr. f. Nervenh. **129**:73 (Dec. 22) 1932.

4. Torrance, A. M.: Meningococcus Meningitis Following Head Injuries, Clin. Med. & Surg. **37**:898 (Dec.) 1930. Korbsch, H.: Meningitis cerebrospinalis epidemica nach Schädeltrauma, Arch. f. Psychiat. **70**:221, 1923-1924. Kalb, A.: Ueber Meningokokkenmeningitis nach Schädeltraumen, Beitr. z. klin. Chir. **124**:211, 1921. Voss, O.: Epidemische Zerebrospinalmeningitis im Anschluss an Schädelbasisfrakturen, Ztschr. f. Laryng., Rhin., Otol. **17**:221 (Dec.) 1928. Lindstrom, E.: Meningitis cerebrospinalis post traumam, Hygiea **5**:708 (July) 1905.

5. Busi, A.: Intorno a un caso di meningite cerebro-spinale traumatica, Arch. di psichiat. **26**:497, 1905. Richey, de W. G., and Helmbold, T. R.: A Case of Meningococcic Meningitis Following Head Injury, Am. J. M. Sc. **166**:559 (Oct.) 1923. Munk, W.: Den traumatiske purulent meningitis, Bibliot. f. læger **119**: 218 (Feb.) 1927. Brändle, U.: Trauma und Meningitis, Dissert., Vienna, Holzworth and Berger, 1932. Jakob, F.: Posttraumatische Meningokokken-Meningitis, Schweiz. med. Wchnschr. **64**:630 (July 7) 1934.

6. Weitzel, L., and Martin, L.: Rechute de méningite cérébro-spinale après un traumatisme crânien, Presse méd. **34**:1211 (Sept.) 1926.

7. In all 4 cases the identification of the micro-organism was determined by fermentation and agglutination reactions.

tional cultures of the spinal fluid in the subsequent week confirmed the presence of meningococci, and although antimeningococcus serum was given intrathecally at daily intervals, death occurred on May 2, sixteen days after the patient's admission to the hospital.

Necropsy revealed a vertical fracture of the occipital bone with extension into the right petrous bone. There were contrecoup lacerations of the brain and subdural hemorrhage. A slight hemorrhage was encountered in the sphenoid sinus, but the ethmoid sinuses and middle ears showed no suppuration. There was an abundant collection of greenish purulent exudate over the vertex and the dorsal surface of the cerebellum.

Case 2.—J. T., a 61 year old white man, was admitted to the psychiatric service on May 16, 1936. The patient was irrational and could give no account of himself. Examination disclosed abrasions over the occipital region of the scalp and contusions over the left frontoparietal area and left thigh. Caked blood was found in the nares. The temperature was 98.6 F., and the spinal fluid was bloody. During the first two days the patient remained confused and disoriented. On May 19 the temperature rose to 103.2 F., and stiffness of the neck and a positive Kernig sign were elicited. The spinal fluid was xanthochromic and contained many polymorphonuclear leukocytes and gram-negative intracellular diplococci, which were identified on culture as meningococci. Antimeningococcus serum was given daily by the intraspinal route, but the patient died on May 22, six days after admission to the hospital.

At neropsy a fracture of the skull was encountered which ran about ¾ inch (1.9 cm.) to the left of the occipital apex down into the foramen magnum and extended up between the vascular process in the occipital portion and the left temporal bone through the sella turcica longitudinally. There was a laceration of the brain on the under-surface of the frontal and temporal lobes on the right side. A leptomeningitis of the purulent type was found around the pons and the cerebellum.

Case 3.—L. D., a 49 year old white man, was admitted to the psychiatric service on May 1, 1936. The patient was found on the street in a stupor, and no history could be obtained. Examination revealed dried blood in the nares and in the left external auditory canal. The temperature was 99.6 F., and the spinal fluid was bloody. During the next ten days the patient appeared rational and was afebrile, though he complained of headache. On the tenth day, because of persistence of the headache and the elevation of the temperature to 104 F., the spinal tap was repeated, and the fluid contained many polymorphonuclear leukocytes and gram-negative diplococci, which were identified on culture as meningococci. A daily spinal tap also showed the fluid to contain meningococci. The patient was daily given intraspinal injections of antimeningococcus serum and appeared to be doing well until acute parotitis developed. He died on June 2, thirty-two days after admission to the hospital.

At necropsy a transverse fissure fracture was noted in the petrous portion of the left temporal bone. The cells of the left middle ear and mastoid contained greenish yellow pus. The air cells of the right ear and mastoid and the right sphenoid and ethmoid sinuses were normal. Reddish brown discoloration of the dura was found over the right parietal and occipital regions. A greenish yellow purulent exudate was encountered in the cisterna magna.

Case 4.—S. Z., a 27 year old professional wrestler, was admitted to the psychiatric service on June 1, 1936. On May 26, while engaged in a wrestling match, he was thrown out of the ring. Shortly thereafter he went to his hotel,

where he fell out of a second story window. No record is available of his course previous to the admission to the hospital, except that he had been disoriented and violent. On admission he appeared quiet and cooperative and presented multiple contusions. The spinal fluid was purulent and contained meningococci in pure culture. Antimeningococcus serum was given intrathecally, but deepening stupor appeared and death occurred forty hours after the patient's admission to the hospital. Culture of the final spinal puncture fluid yielded meningococci and type II pneumococci.

At necropsy a fissure fracture, about 1 cm. in length, was found in the petrous portion of the left temporal bone. Blood was encountered in the right middle ear, but the left middle ear, the mastoid cells and the sphenoid and ethmoid sinuses contained neither blood nor pus. A purulent leptomeningitis was present over the the vertex of the brain.

COMMENT

All 4 patients exhibited the coexistence of fracture of the skull and meningococcic meningitis. In cases 1, 2 and 3 the sequence of events appears clear, in that meningococcic meningitis followed fracture of the skull in five, three and ten days, respectively. In case 4 meningitis was present when the patient was first observed, and the sequence of events is therefore uncertain. It is possible but not probable that the patient had meningitis at the time of the wrestling match.

Although it is to be admitted that these 4 cases and the 16 cases reported in the literature are too few to establish definitely the direct causal relationship between fracture of the skull and meningococcic meningitis, certain considerations support this view. That the meninges may become readily invaded by pathogenic micro-organisms from the nasopharynx after fracture of the skull is well illustrated by the frequent cases of pneumococcic meningitis which occur following fracture of the base of the skull (Vance [8]). Therefore, when meningococci are present in the nasopharynx, it is to be expected that they too may invade and reach the subarachnoid space if the barrier between the nasopharynx and the meninges suffers injury. It is well known that meningococci are present in the nasopharynx of carriers and from time to time in otherwise normal persons.[9] It is also known that the disease

8. Vance, B. M.: Fractures of the Skull: Complications and Causes of Death; Review of Five Hundred and Twelve Necropsies and of Sixty-One Cases Studied Clinically, Arch. Surg. **14**:1023 (May) 1927.

9. (*a*) Albrecht, H., and Ghon, A.: Ueber die Aetiologie und pathologische Anatomie der Meningitis cerebrospinalis epidemica, Wien. klin. Wchnschr. **14**: 984 (Oct.) 1901. (*b*) Zinsser, H., and Bayne-Jones, Stanhope: A Text-Book of Bacteriology with a Section on Pathogenic Protozoa, ed. 7, New York, D. Appleton-Century Company, Inc., 1934, pp. 443-446. (*c*) Rake, G.: Studies on Meningococcus Infection: VI. The Carrier Problem, J. Exper. Med. **59**:553 (May) 1934. (*d*) Tillett, W. S., and Brown, T. M.: Epidemic Meningococcus Meningitis, Bull. Johns Hopkins Hosp. **57**:297 (Nov.) 1935.

may ultimately develop in carriers who have harbored meningococci for some time without symptoms.[10] Hence, it is reasonable to believe that meningococcic meningitis may appear in a carrier when fracture of the skull has created conditions suitable for invasion by the micro-organisms. That 3 of these cases were seen during the recent outbreak of meningococcic meningitis in New York [11] may be but a reflection of the presence of these micro-organisms in the nasopharynx in large numbers of the general population during epidemic periods.

In cases 1 to 3 the possibility is to be considered that the patient acquired the infection after admission to the hospital by contact with meningococcic carriers. Although there were no patients with meningococcic meningitis in the wards to which these patients were admitted, such patients were present in the other wards of the psychiatric medical service at the same time. This possibility, however, does not diminish the responsibility of the fracture of the skull in bringing about the meningitis. Although 30 cases of meningococcic meningitis have been observed in the wards of the psychiatric medical service of the Bellevue Hospital in the last eighteen months, no instance of contact infection of patients or hospital personnel has been observed. This is in accordance with the observations of others [12] on the extreme rarity of obvious infection from contact. However, in the recent outbreak in New York multiple cases of meningococcic meningitis were observed in 4 per cent of the families,[11] and in Baltimore [9d] a history of contact was noted in 7 of 21 cases.

It appears possible from the case reports already cited [13] that trauma may play a predisposing rôle in meningococcic meningitis without fracture of the skull. This receives some support from the experience of those who have observed the development of meningitis caused by the common pyogenic micro-organisms following trauma to the skull without fracture.[14]

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10. MacCallum, W. G.: A Text-Book of Pathology, Philadelphia, W. B. Saunders Company, 1932, pp. 536-537. Zinsser and Bayne-Jones.[9b]

11. Bolduan, C., and Frant, S.: The Recent Increase in Meningococcus Meningitis, Quart. Bull. Dept. of Health, New York **4**:47, 1936.

12. Simon, C. E.: Human Infection Carriers, Philadelphia, Lea & Febiger, 1919, pp. 106-133. Zinsser and Bayne-Jones.[9b]

13. Footnotes 1, 2 and 5.

14. (*a*) Weichselbaum, A., in Kolle, W., and Wassermann, A.: Handbuch der pathologenen Mikroorganismen, Jena, G. Fischer, 1903, vol. 3, p. 293. (*b*) Wechsler, I. S.: A Text-Book of Clinical Neurology, ed. 2, Philadelphia, W. B. Saunders Company, 1931, p. 370. (*c*) Finkelnburg, R.: Die Erkrankungen der Meningen, in Lewandowsky, M.: Handbuch der Neurologie, Berlin, Julius Springer, 1911, vol. 2, p. 1091. (*d*) Ehrnrooth, E.: Eitrige Meningitis, akut entstanden nach Kopftrauma ohne nachweisbare Wunde, Deutsche Ztschr. f. d. ges. gerichtl. Med. **12**:30 (Aug. 27) 1928.

Although in our cases of meningococcic meningitis a potential communication between the meninges and the nasopharynx was created by the fracture of the sphenoid bone in case 2 and of the petrous portion of the temporal bone in cases 1, 3 and 4, it is not known that the micro-organisms traveled along this route. It is possible that trauma to the meninges and the brain furnishes an area of diminished resistance for micro-organisms invading directly from the nasopharynx or from the blood stream. In this connection, Ehrnrooth [14d] has shown in experimental animals that trauma to the head without fracture of the skull promotes the development of meningitis after the intravenous injection of micro-organisms. The rôle played in the propagation and dissemination of the meningococci by otitis and mastoiditis in case 3 and by hemorrhages in the sphenoid sinus in case 1 and in the middle ear in case 4 is not clear.

SUMMARY

Four cases are reported in which meningococcic meningitis followed fracture of the base of the skull. Attention is called to 16 similar cases reported in the literature, and the considerations are discussed which support the view that a direct causal relationship exists between fracture of the skull and meningococcic meningitis.

Drs. Thomas Gonzales, Benjamin Morgan Vance and Milton Helpern, of the Office of the Chief Medical Examiner, New York, supplied the necropsy records.

EFFECT OF STARVATION ON THE HEALING OF FRACTURES IN RABBITS

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Prolonged abstinence from foods providing certain essentials to normal life processes results in various well known deficiency diseases which must be distinguished from changes arising in normal animals suddenly deprived of all food intake. For purposes of clarity the former may be regarded as changes arising from chronic starvation and the latter as those arising from acute starvation. It is with healing in diseases arising from acute starvation that this paper is concerned.

A review of the literature reveals that investigators unanimously agree that acute starvation does not inhibit either growth or repair. McCollum and his co-workers [1] demonstrated that fasting would cause active rickets in rats to heal from within twenty-four to forty-eight hours. Shipley and Holt [2] reported studies in vitro and stated that solutions to which 51.3 millimols of sodium chloride had been added inhibited calcification in preparations of the bones of rats with rickets, while similar preparations from starved rats calcified perfectly. Harvey and his co-workers,[3] who studied the effect of starvation on fibroplasia and the healing of wounds in the stomach of the rat, found that starvation produced no change in the initiation of growth or in its velocity. Roegholt [4] found that lack of vitamin A did not slow up the processes of healing in a wound. Morgan [5] stated that the legs of starved salamanders regenerated after amputation as rapidly as those of normally fed animals.

From the Department of Surgery of the University of Chicago.

1. McCollum, E. V.; Simmonds, N.; Shipley, P. G., and Park, E. A.: The Effect of Starvation on the Healing of Rickets, Bull. Johns Hopkins Hosp. **33**:31, 1922.

2. Shipley, P. G., and Holt, L. E., Jr.: Effect of Starvation on Healing of Rickets in Rats, Proc. Soc. Exper. Biol. & Med. **25**:32, 1927.

3. Howes, E. L.; Briggs, H.; Shea, R., and Harvey, S. C.: Effect of Complete and Partial Starvation on the Rate of Fibroplasia in the Healing Wound, Arch. Surg. **27**:846 (Nov.) 1933.

4. Roegholt, M. N.: Influence of Diet Free from Vitamin A on Myelopoiesis and the White Blood Picture and Its Effect on Wound Healing, Nederl. tijdschr. v. geneesk. **2**:3744 (Aug. 10) 1929.

5. Morgan, Thomas H.: The Physiology of Regeneration, J. Exper. Zoöl. **3**:457, 1906.

There is even some evidence that cell division is stimulated by starvation, as Morgulis and his co-workers [6] quoted Gaglio [7] as finding mitotic figures more frequent in tissues from starved than in those from normal frogs and Morpurgo [8] as finding the same for pigeons. Morgulis concluded from his studies that there is a reduction in cell cytoplasm in starved animals but little reduction in the nucleus. This disturbed ratio results in an increased rate of cell division in an effort to restore the proper balance. Harvey and his co-workers found in the rat that healing of well approximated clean wounds of soft parts is usually complete by fibroplasia in about fourteen days. However, healing of fractures involves more than repair by fibroplasia and requires a longer time. The callus that forms is at first fibrous and subsequently ossifies, partly by fibrous ossification and partly by enchondral ossification.

In order to test the effects of starvation on the phases of the healing of fractures, the following experiments were carried out.

EXPERIMENTAL WORK

Sixty-two adult rabbits of approximately the same age, weight and general physical condition were operated on, and the ulna was fractured by open operation about 1 inch (2.5 cm.) above the joint. No attempt at immobilization of the fracture was made. The animals were then divided into two groups. One group was fed the usual laboratory diet; the other group was placed in metabolism cages so that all excretions were immediately disposed of, and their intake was limited to a 0.9 per cent solution of sodium chloride. There was considerable variation in the ability of the animals to withstand starvation, as they succumbed at intervals ranging from nine to thirty-seven days. Control animals were killed at intervals corresponding in duration to those which succumbed to starvation. The specimens were dissected, roentgenograms were made and the specimens were then sectioned for microscopic examination.

RESULTS

RABBIT 745.—*Eighteen day fracture; normal diet.*

There was a large amount of external callus which was practically entirely ossified. Mesially both the radius and the ulna participated in the process of ossification. In the mesial external callus a large displaced fragment of cortex without nuclei was seen. New bone was being laid down on the dead fragment on two sides. Of the other two sides, the long side adjacent to the fibrous portion of the callus was without new bone, and the end in contact with the radius was necrotic and disintegrating, as was the contacting portion of the radius. The

6. (*a*) Morgulis, S.; Howes, P. E., and Hawk, P. B.: Studies on Tissues of Fasting Animals, Biol. Bull. **28**:397, 1915. (*b*) Morgulis, S.: Fasting and Undernutrition: A Biological and Sociological Study of Inanition, New York, E. P. Dutton & Company, 1923.

7. Gaglio, G.: Sulle alterazioni istologiche e funzionali dei muscoli durante l'inanizione, Arch. per le sc. med. **7**:301, 1884; cited by Morgulis.[6b]

8. Morpurgo, B.: Sull processo fisiologico di neoformazione callulare durante la inanizione acute dell' organismo, Arch. per le sc. med. **12**:395, 1888; cited by Morgulis.[6b]

marked radial periosteal reaction ceased abruptly where contact with the dead fragment began. No cartilage was seen in the lateral external callus and only a small island in the mesial external callus. The fractured ends of the ulnar cortex were shredded and undergoing absorption, as evidenced by the large haversian canals.

The callus separating the opposing fragments of the ulnar cortex was ossified only in that region immediately surrounding the bone, leaving a wide strip of fibrous callus between the two ends. The medullary callus was a dense bony plate with a narrow unossified line in the center extending transversely across the medullary cavity. Numerous displaced spicules of cortical bone were present with new bone laid down on them. There was only a minimal amount of endosteal reaction present, it being most marked on the cortex next to the radius.

RABBIT 686.—*Eighteen day fracture; starvation.*

A thin line of lateral external callus was ossified except in the region of the fracture and a few millimeters of the adjacent cortex. Here there was a dense fibrous callus which separated the ends of the ulna and extended completely across the fracture defect. Disseminated throughout were spicules of cortical bone without nuclei and giant cells, some lying in contact with the dead bone.

There was no callus formation on the mesial side. In the interosseous region, between the ends of the fractured ulna and in the adjacent portion of the medullary cavity, was an acellular granular mass of necrotic tissue, representing the remains of an unabsorbed hematoma. The medullary callus was composed of a wide central band of moderately dense fibrous tissue with many large vacant spaces like those left where fat has been dissolved out during fixation. Peripherally, there were two bands of beginning ossification; the trabeculae here were thin and lacelike, and the cells were larger and closer together than those seen in the specimen from rabbit 745. There was a marked endosteal reaction present. The haversian canals of both the radius and the ulna were much larger than those in the specimen of rabbit 745, indicating osteoporosis.

Comment.—The specimen from the starved animal could be identified microscopically by the lesser amount of external callus, lack of ossification of both the external and the medullary callus, the spongy nature of the ossification present and the presence of osteoporosis.

RABBIT 737.—*Twenty-two day fracture; normal diet.*

The lateral external callus was ossified except for the region between the fragments of the fractured ulna. A small island of cartilage was attached to each of the fragments. The ossification of the mesial callus was limited to the few millimeters adjacent to the cortex of the radius and the ulna. The region immediately adjacent to the fracture line was cartilaginous, and between this cartilage and the fractured ulna were two small pieces of necrotic and disintegrating cortex. The fragments were in good alinement, and in the first few millimeters adjoining the fracture line no nuclei were seen. The region between the two ends contained a small amount of an acellular and granular material, representing an unabsorbed hematoma. On the medullary cavity side of the fracture line was a piece of necrotic disintegrating bone completely surrounded by a large island of cartilage and new bone which spanned the defect.

The medullary callus was ossified peripherally, forming two wide bands of dense bone which stretched transversely across the medullary space and were separated from one another by a narrow band of unossified callus, fibrous in the lateral half and cartilaginous in the mesial half. Spicules of displaced dead cortical

bone with new bone laid down on them still remained. The endosteal reaction and ossification of the callus were more marked mesially than laterally.

RABBIT 681.—*Twenty-two day fracture; starvation.*

The lateral external callus was only partially ossified, being most marked in the cortical region of the proximal fragment. Mesially, ossification of the callus was occurring on the surface of both the radius and the ulna. There was a medullary callus at the site of the fracture which was cartilaginous in the intermediary zone and ossified distal to this in the ends of the fragments. It contained some spicules of dead bone that had been crushed into the cavity by the cutting forceps. Their cells had disappeared, and there was irregular formation of new bone on their surfaces.

Comment.—There was no doubt that both fractures were healing, but that healing was delayed in the starved animal was attested by a lack of ossification of the external callus, relatively less ossification of the medullary callus, lack of endosteal reaction and less actively appearing fibrous callus as compared with the specimen from the normal animal.

RABBIT 679.—*Twenty-nine day fracture; normal diet.*

The callus was completely ossified, forming a dense spongy plate of bone which united the fragments of the ulna. Between the fractured ends an acellular granular precipitate was seen, again representing the remainder of an unabsorbed hematoma. The cortices of the ends of the fragments were without nuclei for a short distance away from the fracture line. The periosteum of the radius in the neighborhood of the fracture had formed a layer of new bone.

The medullary callus was ossified except for one small island of cartilage. The ossified callus formed a wide plate of dense new bone composed of thick wavy trabeculae with small marrow spaces. In the meshes of this new bone lay numerous spicules of dead cortex undergoing bony transformation by creeping substitution.

RABBIT 683.—*Twenty-nine day fracture; starvation.*

The soft tissues attached along the lateral side were densely fibrous and included portions of tendons and muscle. That portion near the fracture line and in the periosteal regions was ossified. A large spicule of dead cortex lay out in the periphery of the lateral callus surrounded by dense fibrous tissue, except on one end. Here there was an island of cartilage with cartilage cells invading the dead spicule. The fragments of the ulna were dead for from 1 to 5 mm. away from the fracture line. A fibrous callus separated the fragments, ossification being limited to that portion immediately about the surface of the fracture.

There was no callus formation on the radial side. Here the fragments were in good alinement, and in the intervening space was a necrotic granular mass of tissue. The fragments were dead in the portions adjoining the fracture line and were undergoing absorption. The medullary callus was separated into two parts by a band of fatty marrow. That part associated with the lateral fragments of the ulna was composed of a central band of dense fibrous tissue continuous with the lateral callus and ossified peripherally where dead spicules of cortex were also being transformed. Here there was only a minimal amount of endosteal reaction. On the radial side a well marked endosteal reaction formed a hillock of new bone, which completely spanned the cortical defect. In the center of

this hillock and spanning the fracture defect lay an island of cartilage. The ossification on the distal fragment was by spongy bone, and here there were many multinucleated giant cells. On the proximal fragment the bony trabeculae were heavier, and only a few giant cells were present.

Osteoporosis of the fragments of both the radius and the ulna was marked. The large number of giant cells present was an outstanding feature.

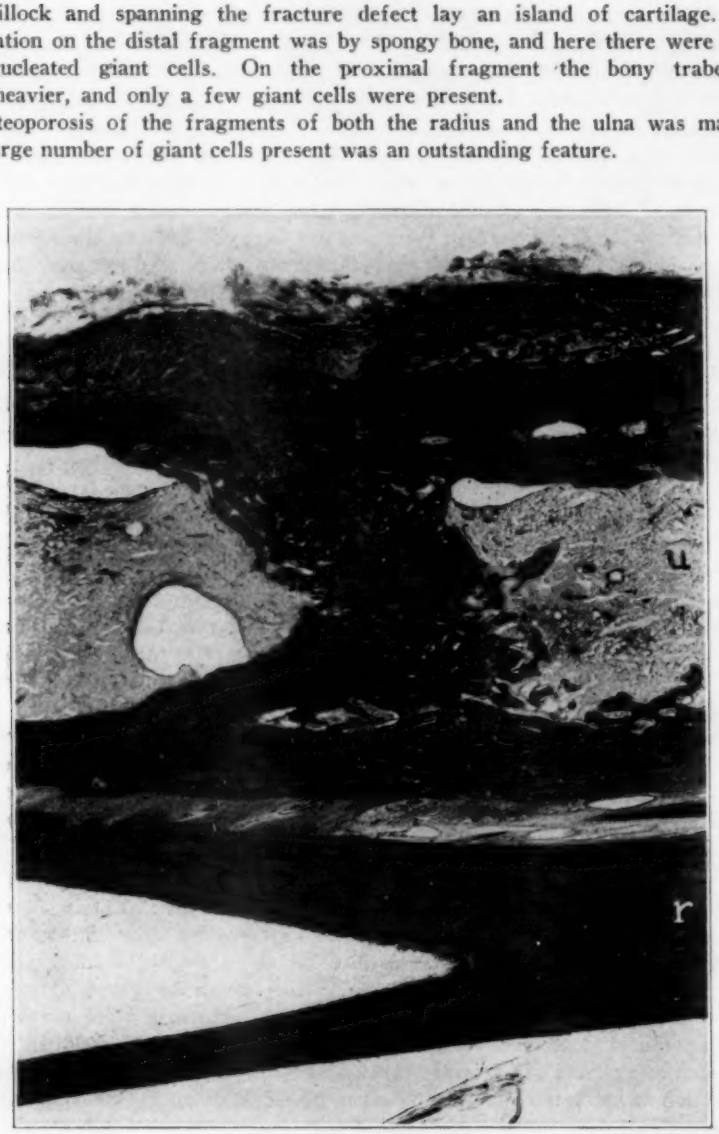

Fig. 1.—Section of a thirty-three day fracture of the ulna (*u*) of a rabbit on a normal diet. The callus is ossified. The radius is indicated by *r*.

Comment.—The specimen from the animal on a normal diet showed bony union, while that from the starved animal showed a callus that was partly ossified, and the bone was more spongy than that of the first specimen. The specimen from the starved animal showed marked osteoporosis.

RABBIT 239.—*Thirty-three day fracture; normal diet.*

A band of almost completely ossified callus united the fragments of the ulna and formed a wide bony bridge, which extended transversely across the medullary canal. A few cartilage cells were present in the intermediary callus. A few fragments of displaced cortical bone without nuclei lay scattered throughout this bony callus and were completely surrounded by living bone which had been

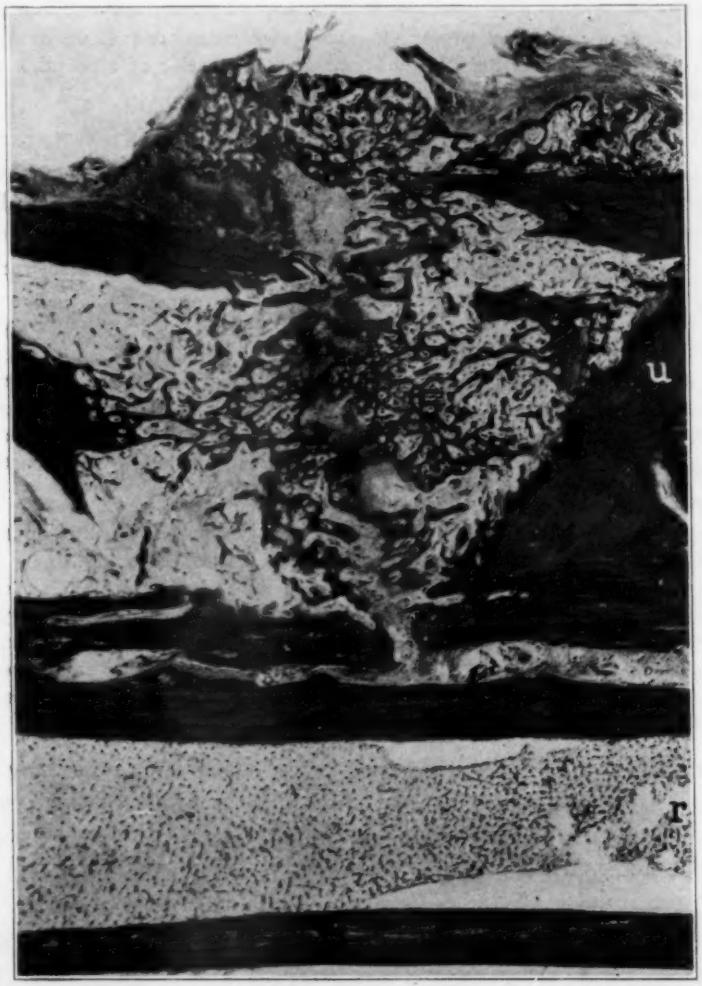

Fig. 2.—Section of a thirty-three day fracture of the ulna of a starved rabbit. The intermediary callus is fibrocartilaginous, and the bony callus is less dense than that in the control specimen (fig. 1). The ulnar (*u*) and radial (*r*) cortexes were osteoporotic.

deposited on them. The mesial cortex was united so well that if it were not for a large piece of dead cortical bone extending at an angle of 45 degrees into the medullary cavity and still attached at one end, one might doubt that a fracture had ever been present.

RABBIT 321.—*Thirty-three day fracture; starvation.*

The lateral callus was ossified by a spongy type of bone. Between the ends of the fragments the fibrous callus remained unossified. There was no mesial callus formation, and the fragments here were not united, the intervening space being filled by tissue like the marrow elsewhere in the specimen. The ossification of the medullary callus was by a spongy type of bone, and extending transversely across the medullary cavity was a band of unossified callus composed about equally of fibrous tissue and cartilage. Marked osteoporosis was present. Giant cells and mononuclear cells were present in the dilated canals and along the surfaces of the cortex and also in the spongy bony callus, indicative of absorption of both the old and the new bone.

Comment.—These two specimens present a sharp contrast, revealing conclusively the inhibiting effect on bony union of acute starvation. The specimen from rabbit 239, the normal animal, shows a healed fracture; that from rabbit 321, the starved animal, shows an ununited fracture with the fracture line wide open, retarded ossification by a spongy bone and marked osteoporosis.

RABBIT 242.—*Thirty-four day fracture; normal diet.*

The ends of the fragments were united by densely ossified callus. The peripheral callus was small and roughly spindle shaped, and the medullary callus extended across the fracture line. It consisted of dense trabeculated bone, and there were a few giant cells and osteoblasts present, indicative of transformative activity.

RABBIT 246—*Thirty-four day fracture; starvation.*

The ends of the fragments were connected by an unossified callus. Practically no periosteal callus was present. The intermediary callus was largely fibrous and the remainder cartilaginous. The endosteal callus was limited in amount and consisted of spongy bone. Disseminated throughout the fibrous intermediary callus were spicules of dead cortical bone undergoing absorption and not associated with any new bone formation. Osteoporosis was again marked. The same absorptive activity of old and new bone by mononuclear and giant cells was seen as in the section from animal 321.

Comment.—Again the difference in the specimens is striking. The specimen from the normal animal showed bony union of the fracture; that from the starved animal showed marked delay in ossification of the callus and marked osteoporosis.

RABBIT 349.—*Thirty-seven day fracture; normal diet.*

The lateral cortex was comminuted. A large spicule of cortical bone without nuclei lay diagonally across the fractured region at an angle of about 35 degrees but united at both ends with the neighboring fragment. New bone was being deposited on it by creeping substitution. There was a completely ossified mesial external callus, and the periosteum of the radius had formed a layer of new bone in the region of the fracture. All that remained of the medullary callus were two long narrow strips of bone without nuclei. They resembled displaced cortical bone which had not been absorbed. The only remaining fibrous callus was in a small region just beneath the dead and angulated cortical fragment previously mentioned. The fracture line on the mesial side could be identified with difficulty.

RABBIT 347.—*Thirty-seven day fracture; starvation.*

The fracture on the radial side was united by a callus that was fairly extensive and consisted of spongy bone, except for two small regions of cartilage in the intermediary zones at the limits of a splinter. On the opposite side and in the region of the medulla the callus was fibrous in its intermediary portion. There was a thin layer of ossified endosteal callus bordering on the intermediary callus. The cortex of the ends of the fragments on this side had been extensively absorbed and partly replaced by new bone, and there was a thin layer of periosteal, spongy bony callus on the proximal fragment.

Comment.—The specimen from the normal animal was healed. That from the starved animal was united mesially by a spongy type of new bone, and laterally there was fibrous union. Ossification of medullary callus was only minimal and again chiefly in conjunction with displaced dead cortical spicules.

COMMENT

In normal adult animals the formation and the destruction of bone are in equilibrium. Fracture of a bone in such an animal, however,

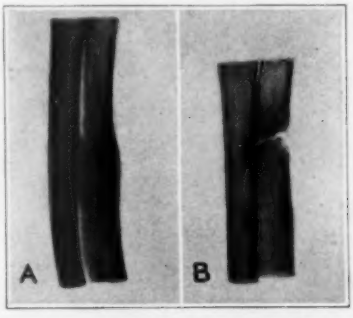

Fig. 3.—Roentgenograms of thirty-four day fractures of the ulna: *A*, a specimen from an animal on a normal diet, and *B*, a specimen from a starved animal.

disturbs the equilibrium locally and causes a shift favoring the formation of bone. Fibroplasia appears in both the periosteal and the endosteal region and proceeds to the formation of a fibrous callus, which later ossifies. The histogenesis of the bone which forms as a result of both enchondral and membranous ossification of the newly formed fibrous callus is complex, for fibroblasts must undergo metaplasia into chondroblasts and osteoblasts, and some of the cartilage is replaced by invading new bone. Complete ossification of the callus eventually takes place.

Suppression of any one or all of these various phases would tend to reverse the equilibrium and thus result in retarded healing time or, if severe enough, lead to nonunion.

Fractures produced in the normally fed rabbits required between three and four weeks for complete ossification of the callus, whereas in none of the fractures in the starved animals was ossification complete, although eleven survived over four weeks. In fact, fractures in

those animals which survived starvation more than thirty days showed less bony callus than did those from animals which succumbed to starvation in from twenty to twenty-six days.

One concludes, therefore, that acute starvation suppresses to some degree the process of ossification of the callus.

Comparison of the specimens from both starved and normally fed animals revealed no obvious difference in the time of appearance or

Fig. 4.—Section of a thirty-four day fracture of the ulna (*u*) of a rabbit on a normal diet. The roentgenogram of this specimen is shown in figure 3 *A*. The callus is ossified. The radius is indicated by *r*.

completion of fibroplasia. Local factors, such as the amount of comminution, approximation of the fragments and disturbance of the periosteum, determine largely the amount of callus formed. Initiation and completion of fibroplasia and the amount of callus formed are therefore not affected by acute starvation. This observation agrees with that of Harvey and his co-workers, who could demonstrate no retarding effect of starvation on fibroplasia in well approximated clean wounds in soft tissue.

Starvation, then, must affect only the process of ossification. This may be accomplished in one or all of three ways, i. e.: by (1) inhibiting the metaplasia of fibroblasts into chondroblasts and osteoblasts; (2) by restricting the bone-forming activity of chondroblasts and osteoblasts to a minimum, or, since the retarding effect was proportional to the duration of the period of starvation, (3) by a shift of the equilibrium

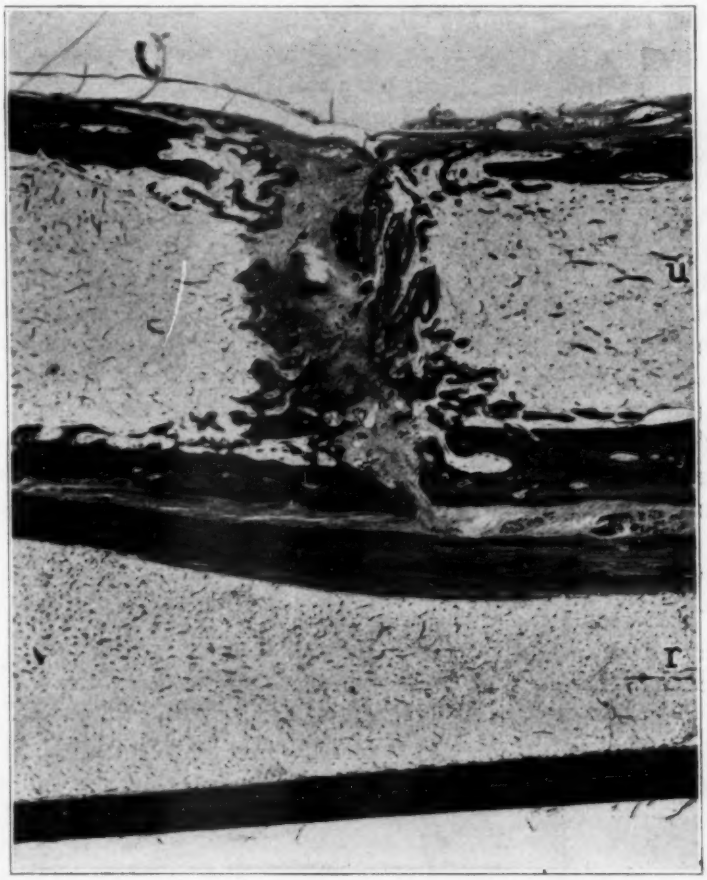

Fig. 5.—Section of a thirty-four day fracture of the ulna (*u*) of a starved rabbit. The roentgenogram of the specimen is shown in figure 3 *B*. The callus is smaller than that in the control specimen (fig. 4). The intermediary callus is fibrocartilaginous, and the bony callus is spongy. The ulnar cortex is markedly osteoporotic, and the radial cortex, less so.

so as to accelerate the destruction of bone at the expense of the formation, in which case one would expect to see a preponderance of osteoclasts resulting in an absorption of the newly ossified callus and a generalized osteoporosis.

Comparison of the specimens from starved and fed animals revealed that metaplasia had occurred in both, for both enchondral and membranous ossification were present. However, the older specimens from the starved animals could be distinguished from those from fed animals by (1) the relatively lesser amount of ossification and (2) the more spongy character of the bone that had been laid down. Generalized osteoporosis was present in the specimens from starved animals, almost from the beginning of ossification of the callus, but was much more marked in the specimens from animals which survived thirty days or more. In addition, the specimens from the latter actually exhibited many osteoclasts, lacunar absorption and less ossification than was seen in specimens from animals succumbing after from twenty to twenty-six days.

One can only surmise as to the actual mechanism of the retarding effect of starvation on ossification of the callus. It would seem, however, that in the beginning starvation chiefly inhibits metaplasia of the fibroblasts and limits the activity of the chondroblasts and osteoblasts to a minimum, so that healing progresses along a fairly normal but definitely inhibited course up to about twenty-six days. Thereafter the constantly increasing shift of equilibrium favoring the destruction of bone (as evidenced by the mild osteoporosis present) seems to be accelerated, osteoporosis increases and osteoclasts and lacunar absorption are seen in the ossified callus.

CONCLUSIONS

1. The healing of a fracture was definitely inhibited by starvation.

2. The process of healing was normal during the stage of the formation of a fibrous callus. But in animals that survived this stage there was definite retardation of ossification of the callus, which became more marked with an increase in the length of the survival period.

3. Generalized osteoporosis gradually developed, and in the animals that survived longest there was some osteoporosis in the callus that had previously ossified.

EFFECT OF ANESTHESIA ON THE BLOOD OXYGEN

II. A STUDY OF THE EFFECT OF SPINAL ANESTHESIA ON THE OXYGEN IN THE ARTERIAL AND IN THE VENOUS BLOOD

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In a preceding communication [1] the existence of anoxic anoxia during ether anesthesia was demonstrated. Spinal or subarachnoid anesthesia is used frequently in surgical procedures, and it is therefore desirable to investigate the effects of spinal anesthesia on the oxygen in the arterial and in the venous blood.

Seevers and Waters [2] stated that the oxygen content in the arterial and in the venous blood is lowered during spinal anesthesia. However, they used large enough doses of drugs to produce respiratory paralysis, which rarely occurs in clinical surgical procedures. They did not present any data to show the degree of change in the blood oxygen. On the other hand, Nowak noted no significant changes in the oxygen of the arterial blood of cats under spinal anesthesia.[3]

In the present investigation we have undertaken to confirm and extend the observations of the previous investigators on the oxygen in the arterial and in the venous blood during a surgical spinal anesthesia.

METHODS

Dogs were used. A lumbar laminectomy was performed two or three days previous to the experiment, to facilitate the performance of a subarachnoid injection of procaine hydrochloride. It was found after several trials of direct injection that this was the most certain method of obtaining spinal anesthesia.

From the Surgical Research Laboratory and the Fifth Surgical (Harvard) Service, the Boston City Hospital.

1. Shaw, J. L.; Steele, B. F., and Lamb, C. A.: The Effect of Anesthesia on the Blood Oxygen: I. A Study of the Effect of Ether Anesthesia on the Arterial and Venous Blood Oxygen, to be published.

2. Seevers, M. H., and Waters, R. M.: Respiratory and Circulatory Changes During Spinal Anesthesia, J. A. M. A. **99**:961 (Sept. 17) 1932.

3. Nowak, S. J. G.: To be published.

The animal was placed in a supine position on the operating board. Under anesthesia with procaine hydrochloride the vena cava was catheterized via the external jugular vein, and the femoral artery was prepared as outlined in a previous communication.[1] After the animal was allowed to become perfectly quiet and adjusted to the surroundings, the pulse and respiratory rates and the blood pressure were recorded. The samples of arterial and of venous blood were taken from the femoral artery and the catheter in the vena cava, respectively.[1] The samples were drawn anaerobically, stored under liquid petrolatum and preserved in an ice bath while the oxygen determinations were being made. Potassium oxalate was used as an anticoagulant.

The animal was then turned on its left side and the laminectomy wound gently opened to expose the dura. This was done without causing the animal any apparent pain or distress. The dog's dura is extremely sensitive, and a puncture cannot be made without producing pain; so before a solution of procaine hydrochloride could be injected into the subdural space it was necessary to administer ethyl chloride for anesthesia for about twenty seconds. While the dog was under ethyl chloride anesthesia a solution of from 10 to 20 mg. of procaine hydrochloride per kilogram of body weight in 1 or 2 cc. physiologic solution of sodium chloride was injected into the subdural space by means of a small needle bent at a right angle. The solution was colored with a few drops of gentian violet in order to make any leakage detectable. Immediately after the injection the ethyl chloride was withdrawn and the animal allowed to recover from the anesthesia. This usually occurred in from sixty to ninety seconds. The animal was then turned back to the supine position. Anesthesia usually began immediately, and the level was noted by testing the skin with forceps or a sharp instrument.

Samples of arterial and of venous blood were taken, and the blood pressure and the pulse and respiratory rates determined after periods of thirty minutes and one hour, respectively. The animals were killed at the end of the experiment by quick intravenous injection of 100 mg. of sodium amytal per kilogram of body weight. Death occurred almost instantly. The trachea was exposed and clamped, and an immediate postmortem examination of the lungs was made.[4]

Blood oxygen determinations were made by the technic of Van Slyke and Neill.[4]

RESULTS

Eight experiments were performed. The results of the blood oxygen determinations for the experiments are recorded in tables 1 and 2 and shown graphically in charts 1 and 2.

The oxygen capacity of the arterial blood showed equivocal changes, though it tended to show changes of greater magnitude when decreasing.

In five of the eight experiments there was an increase in the oxygen content of the arterial blood. The greatest increase was 8.5 per cent, and the greatest decrease was 15.2 per cent. The mean change for the group was a slight increase.

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4. Van Slyke, D. D., and Neill, J. M.: The Determination of Gases in Blood and Other Solutions by Vacuum Extraction and Manometric Measurement, J. Biol. Chem. **61**:523. 1924.

TABLE 1.—*Oxygen Values in Arterial and in Venous Blood of Dogs Under Spinal Anesthesia*

Experiment No.	Oxygen Capacity of Arterial Blood			Oxygen Content of Arterial Blood			Oxygen Content of Venous Blood			Arteriovenous Difference			Oxygen Saturation of Arterial Blood*			Oxygen Saturation of Venous Blood*		
		Blood Sample After Anesthesia			Blood Sample After Anesthesia			Blood Sample After Anesthesia			Blood Sample After Anesthesia			Blood Sample After Anesthesia			Blood Sample After Anesthesia	
	Control Blood	30 Min.	1 Hour	Control Blood	30 Min.	1 Hour	Control Blood	30 Min.	1 Hour	Control Blood	30 Min.	1 Hour	Control Blood	30 Min.	1 Hour	Control Blood	30 Min.	1 Hour
	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Volumes per Cent	Per-centage	Per-centage	Per-centage	Per-centage	Per-centage	Per-centage
2	24.9	26.4	26.2	23.0	25.2	24.8	17.1	18.6	17.4	5.9	6.6	7.4	92.4	95.5	94.6	68.8	70.5	66.4
3	16.0	15.5	15.4	13.4	13.9	14.3	8.8	4.8	3.8	4.6	9.1	10.5	84.0	89.7	92.9	55.0	30.9	24.7
5	17.7	16.7	17.2	16.2	16.0	15.9	11.1	7.9	8.2	5.1	8.1	7.7	91.5	95.8	92.4	62.7	47.3	47.6
6	22.6	23.6	22.8	21.1	21.4	20.9	10.7	12.2	11.5	10.4	9.2	9.4	93.5	90.7	91.7	47.5	51.8	50.4
7	20.8	17.9	17.6	19.0	16.8	16.1	15.6	6.9	5.9	3.4	9.9	10.2	91.3	93.8	91.5	75.0	38.7	33.5
8	19.7	17.4	17.7	17.9	17.3	17.2	12.0	4.3	9.6	5.9	13.0	7.6	90.4	99.4	97.2	61.0	24.6	54.3
10	16.2	16.5	16.6	14.4	15.0	14.5	9.8	8.0	...	4.6	7.0	...	88.9	90.7	87.3	60.5	48.5	....
11	13.9	14.4	14.1	12.8	13.8	13.9	9.2	7.2	7.6	3.6	6.6	6.3	92.2	95.8	98.5	66.2	50.0	53.9
Mean percentile change for the experimental series		−2.31	−2.71		+1.31	+0.47		−28.14	−26.65		+78.12	+75.92		+3.47	+2.83		−16.7	−15.05
Mean variation of the mean percentile change †		±4.39	±5.69		±5.78	±7.44		±19.50	±21.30		±59.28	±58.75		±2.35	±3.60		±13.17	±14.51
Mean of the mean variation of the mean percentile change		±1.55	±2.01		±2.05	±2.63		± 6.89	± 7.53		±22.43	±20.76		±0.83	±1.21		± 4.65	± 5.14

* In the computation of the oxygen saturation of the arterial and venous blood, 0.5 and 0.3 volumes per cent were deducted from the oxygen capacity of the arterial blood and the oxygen content of the arterial and venous blood, respectively, to correct for the dissolved oxygen not in combination with the hemoglobin. The oxygen capacity of the arterial blood was used to calculate the oxygen saturation of the venous blood for general comparison, since the venous oxygen capacity of the venous blood was not ascertained in all instances.

† Scott, E. L.: What Constitutes an Adequate Series of Physiological Observations? J. Biol. Chem. **73**: 81, 1927.

In seven of the eight experiments there was a decrease in the oxygen content of the venous blood, the greatest decrease being 64 per cent. In one experiment there was an increase of 14 per cent in the oxygen content.

The arteriovenous difference increased sharply in seven of the eight experiments. In the one experiment in which no increase was recorded the initial arteriovenous difference was abnormally high.

In seven of the eight experiments an increase in the oxygen saturation of the arterial blood was noted. The greatest increase noted was 9 per cent and the smallest 0.4 per cent. In a single experiment a decrease in saturation of 2.8 per cent was recorded.

TABLE 2.—*Oxygen Values in Blood Samples Taken from Different Regions of the Body* (Experiment 8)*

Time of Taking Blood Sample	Blood Oxygen	Site of Blood Sample: Femoral Artery	Right Side of Heart†	Femoral Vein	External Jugular Vein
Control	Content (volumes per cent).....	17.9	12.0	11.1	12.4
	Capacity (volumes per cent).....	19.7	20.3		
	Percentage of saturation.....	91.0	61.0	56.4	62.8
30 minutes	Content (volumes per cent).....	17.3	4.3	8.2	7.9
	Capacity (volumes per cent).....	17.4	18.6		
	Percentage of saturation.....	99.7	24.6	47.4	45.7
1 hour	Content (volumes per cent).....	17.2	9.6	10.0	8.9
	Capacity (volumes per cent).....	17.7	19.1		
	Percentage of saturation.....	97.7	54.3	56.4	50.5

* The oxygen capacity of the arterial blood was used in computing the oxygen saturation of the venous blood for general comparison, since oxygen capacity of the venous blood was not determined in all the experiments.

† This sample of blood was taken from the junction of the superior and the inferior vena cava, as outlined under "Methods." It is not truly blood from the right side of the heart, but for practical purposes it should be almost identical.

In six of the eight experiments there was a decrease in the oxygen saturation of the venous blood. In two experiments small increases in the oxygen saturation were recorded, the largest of which was 4.3 per cent.

Table 2 shows the values for the samples of blood taken from different sources. The blood from the anesthetized area shows a slightly higher oxygen content than that taken from the upper part of the body. The difference is not marked and is well within the range of experimental variation.

There was no change in the blood pressure in experiment 5. In experiment 7 there was an increase in the blood pressure of 5.6 and 7.5 per cent for the thirty minute and one hour periods, respectively. Changes in the blood occurred in these experiments in spite of the

failure of the blood pressure to drop. The average changes for the five experiments were 37.5 and 31.1 per cent for the thirty minute and one hour periods, respectively. The lowest blood pressure reached was 40 mm. of mercury, a decrease of 71 per cent from the control.

The respiratory rate showed equivocal changes, though it tended to increase slightly and usually became shallower and largely diaphragmatic.

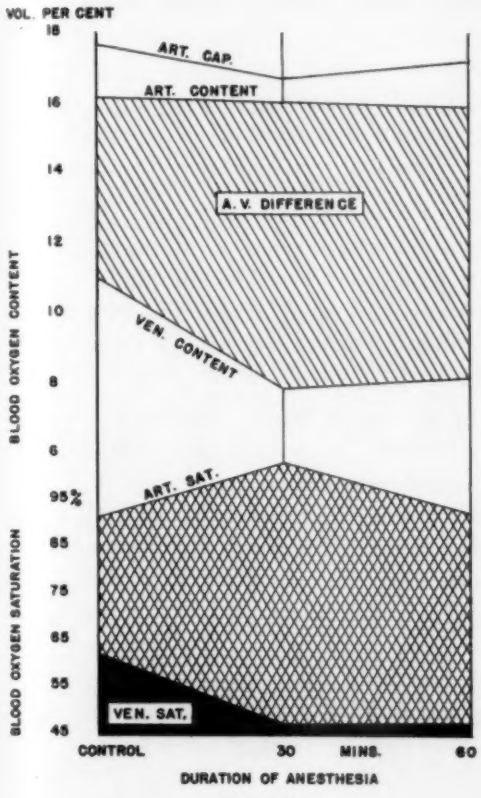

Chart 1

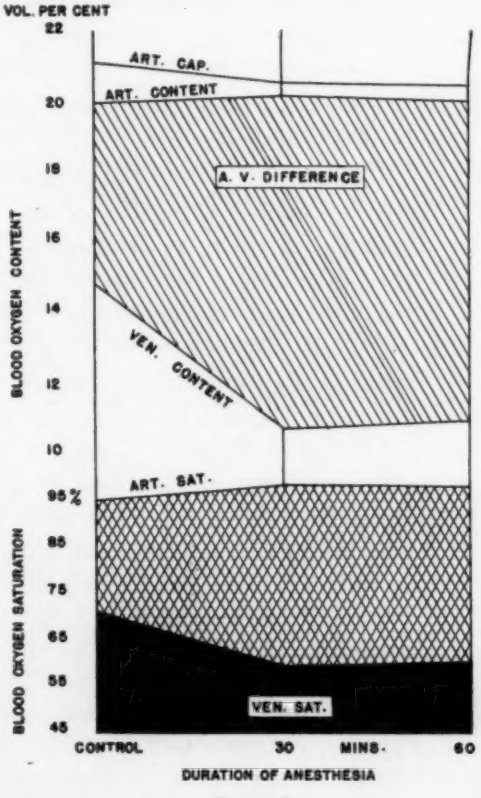

Chart 2

Chart 1.—Blood oxygen determinations in experiment 5.

Chart 2.—Mean of the results of the experimental series. The values used in this chart were calculated by use of the mean percentile changes for the experimental series, and as the control the following normal values for human blood were used: oxygen capacity of the arterial blood, 20.9 volumes per cent; oxygen content of the arterial blood, 19.9 volumes per cent; oxygen content of the venous blood, 14.9 volumes per cent; arteriovenous difference, 5 volumes per cent; oxygen saturation of arterial blood, 95 per cent, and oxygen saturation of venous blood, 71.4 per cent.

On postmortem examination the lungs appeared well inflated and evenly colored, except for a few small areas in the most dependent portions of the bases of the lungs, which were bright red. The striking feature about the lungs was their unusual paleness—a whitish pink—as if they were almost drained of their normal blood content. Removal of the tracheal clamp allowed the lungs to collapse without any areas of diminished crepitation. Gross section showed nothing remarkable, and no fluid could be expressed from the cut surface. The results of the microscopic examination were negative except for sparsely filled capillaries.

COMMENT

It is apparent that spinal anesthesia in the dog produces the following changes:

1. An increase in the oxygen saturation of the arterial blood. This change may be explained by the finding of a diminished content of the pulmonary blood, as indicated by the postmortem paleness of the lungs and a prolonged time for circulation of pulmonary blood.[5] The diminished flow of pulmonary blood plus the exposure of a smaller quantity of blood than normal to the entire capillary-alveolar bed of the lungs insures the complete oxygenation of the arterial blood.

2. A decrease in the oxygen content of venous blood. This change is due to stasis and is found in such conditions as shock[6] and cardiac decompensation. The blood from the right side of the heart may be considered as coming from two sources: the upper, unanesthetized, and the lower, anesthetized, area of the body. Its blood content will be the resultant of the mixture of blood from these two sources. Determinations on the oxygen in blood specimens taken simultaneously from each area of the body should give the source of the stasis. The results in experiment 8, shown in table 2, demonstrate that stasis occurs in both these regions of the body and appears to be slightly greater in the upper, unanesthetized, portion. The low oxygen content of venous blood in the upper, unanesthetized, part of the body is due to the reduced blood flow resulting from a compensatory vasoconstriction necessary to maintain a normal blood pressure. In conformity with the conception of an increased flow of blood through the anesthetized portion of the body due to the vasodilatation from vasoconstrictor

5. Nowak, S. J. G.: Personal communication to the authors. Shaw, J. L.; Steele, B. F., and Lamb, C. A.: Unpublished data on dogs using the sodium cyanide method.

6. Aub, J. C., and Cunningham, T. D.: Studies in Experimental Traumatic Shock: II. The Oxygen Content of the Blood, Am. J. Physiol. **54**:408, 1920-1921.

paralysis,[7] it would be expected that the oxygen content of the venous blood would be normal or elevated. On the contrary, it was observed to be lowered. The vasodilatation in the anesthetized area of the body, unlike the arteriolar dilatation seen when ether anesthesia is used,[8] involves the arterioles, capillaries and venules as a result of complete paralysis of their innervation. Since Krogh[9] has shown that under normal circumstances only a small proportion of the capillaries are used at any one time, this vasodilatation, assisted by the paralysis of the voluntary muscles, enlarges the vascular bed and results in the shifting or pooling of part of the blood volume in the anesthetized part of the body. Bradshaw[10] has shown that this vasoconstrictor paralysis is responsible for the fall in blood pressure observed in spinal anesthesia. Burch and Harrison[11] have demonstrated that as a sequel to a fall of 30 per cent or more in blood pressure the return of venous blood to the right side of the heart becomes inadequate, and a decreased cardiac output follows.

It is evident in the animals showing a fall in blood pressure that the pooling of blood from the vasodilatation in the region of anesthesia plus a diminished cardiac output and lowered blood pressure may produce a stasis of sufficient magnitude to explain the low oxygen content of the venous blood. The pale, blanched-appearing lungs may be further evidence of poor venous return to the heart from peripheral pooling.

The findings in the blood from the right side of the heart in the two experiments in which there was an increase and no change, respectively, in the blood pressure could be explained by a generalized vaso-

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7. White, J. C.: Diagnostic Novacaine Block of the Sensory and Sympathetic Nerves: Method of Estimating Results Which Can Be Obtained by Their Permanent Interruption, Am. J. Surg. **9**:264, 1930. Morton, J. J., and Scott, W. J. M.: The Measurement of Sympathetic Vasoconstrictor Activity in the Lower Extremities, J. Clin. Investigation **9**:235, 1930. White, J. C.: Diagnostic Blocking of Sympathetic Nerves to the Extremities with Procaine: Test to Evaluate the Benefit of Sympathetic Ganglionectomy, J. A. M. A. **94**:1382 (May 3) 1930. Burch, J. C., and Harrison, T. R.: The Effect of Spinal Anesthesia on Arterial Tone, Arch. Surg. **22**:1040 (June) 1931.

8. Embley, E. H.: Action of Ether upon the Circulation, Biochem. J. **4**:79, 1910.

9. Krogh, A.: The Anatomy and Physiology of Capillaries, New Haven, Conn., Yale University Press, 1922.

10. Bradshaw, H. H.: The Fall in Blood Pressure During Spinal Anesthesia, Ann. Surg. **104**:41, 1936.

11. Burch, J. C., and Harrison, T. R.: Effect of Spinal Anesthesia on Cardiac Output, Arch. Surg. **21**:330 (Aug.) 1930.

constriction.[12] However, that would be expected to elevate the blood pressure more than the observed 7.5 per cent. Since a flaccid paralysis of voluntary muscles occurred in all instances, the blood pressure may have been maintained either by an adequate vasoconstriction in the unanesthetized area of the body or by a partial generalized vasoconstriction in the anesthetized part of the body. In the two experiments in which there was no change in the blood pressure it may be that the vasodilatation opens up such a vast additional capillary bed through which the blood flow is distributed that, in spite of an adequate blood pressure, the volume of flow through any one channel is diminished. Since the tissue metabolism is not hindered as in ether anesthesia,[13] the oxygen is removed in a greater than normal quantity during this paradoxical stasis. This may not be in accord with the finding of an elevated oxygen content of the venous blood in sympathectomized animals.[14] However, in these animals there had been time for some compensation from the acute vasodilatation, and normal muscular tone and function were present to aid in the movement of blood toward the heart.

3. An increase in the arteriovenous difference. This is the consequence of the lowered oxygen content of the venous blood.

4. Stagnant anoxia. The findings of a perfectly saturated arterial blood and a lowered oxygen content of the venous blood, or an increased arteriovenous difference, are typical of a stagnant anoxia.[15] The stagnant anoxia of spinal anesthesia differs from the anoxic anoxia of ether anesthesia in that the blood oxygen reaches the tissues at an adequate available tension but in an insufficient quantity for the requirements of the tissue metabolism. The results indicate that the stagnant anoxia is general and will vary in degree with the extent of the area anesthetized or the magnitude of the vasoconstrictor paralysis and vasomotor compensation. The significance of anoxia in surgical anesthesia has been discussed in a previous communication.[1]

SUMMARY

Spinal anesthesia in dogs causes the following changes: (1) an increase in the oxygen saturation of the arterial blood, a decrease in

12. Freeman, N. E.: Hemorrhage in Relation to Shock: Experimental Effect of Intravenous Injections of Saline, Gum Acacia, and Blood on the Rate of Adrenal Secretion Resulting from Hemorrhage, Ann. Surg. **101**:484, 1935. Aub and Cunningham.[6]

13. Brow, G. R., and Long, C. N. H.: Biochemical Changes in the Heart During Anesthesia, Anesth. & Analg. **9**:193, 1930.

14. Freeman, N. E.: Personal communication to the authors.

15. Barcroft, J.: Anoxaemia, Lancet **2**:485 (Sept. 4) 1920.

the oxygen content and saturation of the venous blood, and an increase in the arteriovenous oxygen difference and (2) pale, blanched-appearing lungs, possibly indicating incompletely filled lung capillaries.

CONCLUSION

The foregoing changes indicate the existence of a stagnant anoxia. It is suggested that the anoxia may be minimized by the use of small doses of the anesthetizing drug or by controlling the administration in such a way as to involve a minimum number of the sympathetic vasoconstrictor fibers and employing vasoconstricting drugs which act principally on the capillary walls.

EXPERIMENTAL CEREBRAL TRAUMA

THE FLUID CONTENT OF THE BRAIN AFTER TRAUMA TO THE HEAD

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On no subject is there greater divergence of opinion than on the proper methods of treatment of injuries of the head with their resultant cerebral damage. Fay [1] advocated an extreme degree of dehydration by the use of hypertonic solutions, both intravenously and by bowel, and rigid restriction of the fluid intake; Munro [2] advised reduction of pressure and removal of blood by frequent spinal punctures; Sachs [3] expressed the opinion that spinal puncture is dangerous and contraindicated, and Dandy [4] advised against the use of both hypertonic solutions and spinal puncture, preferring operative decompression in cases in which there is evidence of a high increase in the intracranial pressure.

The reason for the contradictory nature of these opinions is not difficult to find. It is based on ignorance of the mechanism of the cerebral damage attending injuries of the head. The literature abounds with postmortem statistics and clinical opinions which attribute the cause of symptoms following injuries to the head to petechial hemorrhage, molecular displacement, edema (both local and general), "traumatic hydrocephalus" and other hypothetic disorders. But there is little positive proof of the existence of any of them.

Adequate experimental studies on the subject are meager. In 1894 Halliburton [5] first called attention to the physiologic significance of the

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1. Fay, T.: Head Injuries: Result Obtained with Dehydration in Forty-Eight Consecutive Cases, J. Iowa M. Soc. **20**:447, 1930; Generalized Pressure Atrophy of the Brain, Secondary to Traumatic and Pathologic Involvement of Pacchionian Bodies, J. A. M. A. **94**:245 (Jan. 25) 1930; The Treatment of Acute and Chronic Cases of Cerebral Trauma, by Methods of Dehydration, Ann. Surg. **101**:76, 1935.

2. Munro, D.: The Diagnosis, Treatment and Immediate Prognosis of Cerebral Trauma: An Introductory Study of 1,494 Cases, New England J. Med. **210**:287 (Feb. 8) 1934; The Therapeutic Value of Lumbar Puncture in the Treatment of Cranial and Intracranial Injury, Boston M. & S. J. **193**:1187 (Dec. 24) 1925.

3. Sachs, E.: The Diagnosis and Treatment of Head Injuries, J. A. M. A. **81**:2159 (Dec. 29) 1923; Head Injuries, Internat. J. Med. & Surg. **46**:567, 1933.

4. Dandy, W. E.: Diagnosis and Treatment of Injuries of the Head, J. A. M. A. **101**:772 (Sept. 2) 1933.

5. Halliburton, W. D.: The Proteids of the Nervous System, J. Physiol. **15**:90, 1894.

fluid content of the brain and pointed out the marked difference between the gray and the white matter in this respect. His findings and conclusions were reiterated in a later paper.[6] In studies on the brains of men, monkeys, dogs and cats he found the average fluid content of the normal gray matter to be 83.5 per cent, and that of the white matter, 69.9 per cent. In 1896 there appeared a monograph by Leonard Hill on "The Cerebral Circulation,"[7] in which he identified cerebral edema as fluid squeezed out of capillaries by some already existing cerebral compression. Shortly thereafter, Courtney[8] suggested that a vasomotor paralysis resulting from the original injury to the head gave rise to arteriolar dilatation and subsequently to edema. In 1901 Cannon[9] summarized existing opinions and reported experiments on trauma to the head, adding hypothetic anemia and osmotic changes to the possible causes of cerebral edema. Liesegang and Mayr[10] in 1912 attributed cerebral edema to accumulation of acid resulting from anemia of the brain.

In these early papers and in nearly all subsequent studies edema of the brain has been assumed to be the principal compressing factor in those cases of injury to the head in which there was no gross hemorrhage or major bony depressions. Some authors, particularly in France and Germany, have postulated the existence of local edema about the third ventricle (Arnaud[11]), the aqueduct (Denis,[12] Karitzky[13]) or the outlet of the fourth ventricle (Hauptmann[14]), with a resulting obstructive hydrocephalus.

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6. Halliburton, W. D.: Die Biochemie der peripheren Nerven, Ergebn. d. Physiol. **4**:23, 1905.

7. Hill, L.: The Physiology and Pathology of the Cerebral Circulation, London, J. & A. Churchill, 1896.

8. Courtney, J. W.: Traumatic Cerebral Edema: Its Pathology and Surgical Treatment; a Critical Study, Boston M. & S. J. **140**:345, 373 and 397, 1899; quoted by Cannon.[9b]

9. Cannon, W. B.: (*a*) Intracranial Pressure After Head Injuries, Boston M. & S. J. **145**:158, 1901; (*b*) Cerebral Pressure Following Trauma, Am. J. Physiol. **6**:91, 1901-1902.

10. Liesegang, R., and Mayr, E.: Hirnschwellung: I. Die Physik und Chemie der Hirnschwellung, Ergebn. d. Neurol. u. Psychiat. **2**:157, 1912.

11. Arnaud, M.: Recherches sur les hypertensions intracraniennes bloquées du liquide céphalo-rachidien consécutives aux traumatismes craniens, et en particulier sur les hypertensions intraventriculaires, Bull. et mém. Soc. nat. de chir. **59**:843 (June 3) 1933.

12. Denis, R.: Au sujet du traitement des lésions traumatiques cranio-encéphaliques, J. de chir. **42**:873, 1933.

13. Karitzky, B.: Hirndruck bei stumpfen Kopfverletzungen, Deutsche Ztschr. f. Chir. **242**:1, 1933.

14. Hauptmann: Die Objektivierung postcommotioneller Beschwerden durch das Encephalogramm, Zentralbl. f. d. ges. Neurol. u. Psychiat. **48**:846, 1927-1928.

Most of the meager experimental studies on the subject of cerebral edema are subject to various criticisms. Apfelbach [15] determined the fluid content of blocks of traumatized brains removed at autopsy but apparently did not separate the gray from the white matter or determine the proportions of each in the blocks. Since the gray and the white matter may differ 10 per cent or more in their water contents, Apfelbach's values cannot be considered significant. The same objection applies to studies on heat stroke by McKenzie and LeCount,[16] as does also the objection that the studies were made at varying and often long intervals after death. The dogs' brains studied by Masserman and Schaller [17] were dried only twenty-four hours at an unstated temperature. Histologic evidence of edema of the brain (Winkelman and Eckel,[18] Masserman and Schaller,[17] Rand and Courville [19] and Hoff [20]) cannot be considered conclusive on account of the many variable factors in the preparation of the material, such as fixation, dehydration, cutting and staining.

There have been no direct experimental studies on the fluid contents of traumatized brains. This fact, as well as many other shortcomings in existing knowledge of the mechanism of injuries to the head, has been recently emphasized in the comprehensive and critically written review of Lehman and Parker.[21] The reader is referred to this paper and to those of Masserman [22] and Winkelman and Eckel [18] for detailed bibliographies.

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15. Apfelbach, C. W.: Studies in Traumatic Fractures of the Cranial Bones: I. Edema of the Brain; II. Bruises of the Brain, Arch. Surg. **4**:434 (March) 1922.

16. McKenzie, P., and LeCount, E. R.: Heat Stroke, with a Second Study of Cerebral Edema, J. A. M. A. **71**:260 (July 27) 1918.

17. Masserman, J. H., and Schaller, W. F.: Intracranial Hydrodynamics: II. Influence of Rapid Decompression of the Ventriculosubarachnoid Spaces on the Occurrence of Edema of the Brain, Arch. Neurol. & Psychiat. **30**:107 (July) 1933.

18. Winkelman, N. W., and Eckel, J. L.: Brain Trauma: Histopathology During the Early Stages, Arch. Neurol. & Psychiat. **31**:956 (May) 1934.

19. Rand, C. W., and Courville, C. B.: Histologic Changes in the Brain in Cases of Fatal Injury to the Head: Reaction of Microglia and Oligodendroglia, Arch. Neurol. & Psychiat. **27**:605 (March) 1932.

20. Hoff, H.: Experimentelle Studien zur Frage des postkommotionellen Hirnödems, Ztschr. f. d. ges. Neurol. u. Psychiat. **129**:583, 1930.

21. Lehman, E. P., and Parker, W. H.: The Unsolved Problems of Brain Injury: A Critical Review of the Literature, Internat. Clin. **3**:181, 1935.

22. Masserman, J. H.: Intracranial Hydrodynamics: Central Nervous System Shock and Edema Following Rapid Fluid Decompression of the Ventriculo-Subarachnoid Spaces, J. Nerv. & Ment. Dis. **80**:138, 1934; Cerebrospinal Hydrodynamics: IV. Clinical Experimental Studies, Arch. Neurol. & Psychiat. **32**: 523 (Sept.) 1934. Masserman and Schaller.[17]

The experiments to be reported herein were designed to determine the effect of trauma to the head on the fluid content of various portions of the brain and on the volume outflow of cerebrospinal fluid. In other experiments the effect of the intravenous administration of hypertonic solution of dextrose in traumatized animals was determined.

METHODS AND RESULTS

All of the sixty experiments were performed on dogs weighing from 7 to 12 Kg. The animals were anesthetized with sodium barbital, 0.3 Gm. per kilogram of body weight, administered intravenously at least two hours before the beginning of the experiments. The animal's head was held rigidly in a constant position throughout the experiment. Cerebrospinal fluid pressure was determined by means of a needle in the cisterna magna connected with a water manometer. Arterial blood pressure was recorded by means of a cannula in the femoral artery.

Measured trauma was administered to the head by dropping a standard 500 or 1,000 Gm. metal weight on the vertex of the head from a constant height of 60 inches (154 cm.). In each experiment the weight was dropped twice in rapid succession.

After the conclusion of one of the various types of procedure to be described, each experiment was terminated by stabbing the heart. The brain was immediately removed (requiring seven minutes or less), and specimens of gray and of white matter from the cerebral cortex and of gray matter from the cerebellum and a portion of the medulla and pons were quickly separated, placed in weighing bottles or beakers and weighed. Gray matter and white matter were separated by a standard rapid technic, by means of which a minimum of one type of tissue was left clinging to the specimen of the other. A sharp knife was used, and great care was exercised to avoid compressing or macerating the specimens. The constancy of the findings to be related attests the accuracy of the separation.

Specimens were dried to constant weight in an oven at 60 C. (usually requiring four or five days), and their fluid and solid contents were thus determined.

Some animals showed extravasation of blood into the scalp or temporal muscle, and an occasional small laceration of the scalp was produced, but in no instance was either fracture of the skull or perceptible intracranial hemorrhage produced by the trauma. One animal died five minutes after the trauma without showing elevation of the cerebrospinal fluid pressure or of the blood pressure. In this dog the fluid contents of the several specimens from the brain were within the normal range. The results of this experiment are not included in the tables.

In the normal dog, with the head in the position used throughout these experiments, the cerebrospinal fluid usually rose to the top of the needle and occasionally dripped out slowly. It did not rise sufficiently high to become visible in the manometer, to which the needle was attached by an adapter and a short rubber tube. The distance from the needle to the zero point of the manometer was 2 cm. The pressure was considered normal when the fluid was not visible in the manometer, and this normal pressure is designated in the tables by the symbol $\pm$. This degree of pressure ($\pm$) was observed in all animals before the infliction of the trauma.

The various types of experiments will be considered separately, and average findings in all groups of dogs will later appear in the composite table 7.

GROUP 1.—*Normal Controls.*—Five dogs were killed twelve hours after receiving the usual dose of barbital and being kept in the same position as the animals in the remaining experiments. Table 1 shows the fluid contents of the portions of their brains examined. The relatively narrow limits of variation are clearly shown. The values found correspond closely to those found by Calhoun and myself [23] by the same method in twenty-four laboratory dogs which had been previously used for various experiments not involving the nervous system. In

TABLE 1.—*Percentage of Water in the Brains of Normal Dogs Anesthetized with Sodium Barbital*

Dog No.	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
T7	79.9	68.8	79.8	70.4
T14	79.8	67.0	80.0	70.2
T24	79.2	68.2	79.6	70.8
T44	78.3	68.8	79.7	71.9
T45	79.8	67.4	79.2	71.1
Averages	79 4	68.0	79.6	70.9

TABLE 2.—*Fluid Content of the Brains of Dogs Killed at Arbitrary Intervals After Trauma by a 500 Gm. Weight*

Dog No.	Time Dog Was Killed, Hours After Trauma	Immediate Rise in Mean Blood Pressure, Mm. of Hg	Later Rise in Mean Blood Pressure, Mm. of Hg	Cerebrospinal Fluid Pressure: Maximum Increase, Mm. of H_2O	Cerebrospinal Fluid Pressure: Increase at End of Experiment, Mm. of H_2O	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
T1	2	0	0	±	±	82.3	71.0	82.2	74.0
T2	2	5	20	30	30	79.8	69.5	80.5	70.0
T3	2	10	0	20	20	79.5	67.8	80.1	70.0
T4	2	8	0	±	±	79.7	66.0	80.1	69.3
T5	2	0	0	±	±	79.3	68.5	80.3	71.6
T6	4	3	10	35	35	81.0	68.8	80.7	71.2
T51	4	6	10	30	±	79.4	68.2	80.5	70.1
T8	8	0	14	40	±	80.8	70.6	81.2	71.8
T9	8	0	8	20	10	80.1	70.4	80.9	70.2
T10	24	0	0	10	±	79.7	69.5	80.5	70.5
T54	24	0	10	25	5	79.9	69.8	80.4	70.6
T56	24	4	16	35	20	79.5	69.6	80.2	70.2
Average						80.1	69.1	80.6	70.8

three of these animals the cerebrospinal fluid pressure was observed throughout the experiments and was never found to rise above the normal level. Similarly, the mean blood pressure maintained a rather constant level, tending to fall slightly after about eight hours.

GROUP 2.—*Trauma by a 500 Gm. Weight; Animals Killed After Various Intervals.*—Five of the animals in this group were killed two hours after trauma, and two were killed four hours, two eight hours and three twenty-four hours after trauma. The results are shown in table 2.

23. Pilcher, C., and Calhoun, J. A.: Unpublished observations.

An increase in the cerebrospinal fluid pressure within two hours was produced in only two of the five dogs, and the increase in pressure did not exceed 40 mm. of water in any of the twelve animals. A typical curve of cerebrospinal fluid pressure after this degree of trauma is shown in chart 1.

Changes in blood pressure were not marked in any animals of this group.

The fluid content of the various portions of the brain showed small variations in individual instances, but the average values indicate a small but definite increase in the fluid content of the cerebral and the cerebellar gray matter, beginning two hours after the trauma and increasing until the eight hour period, followed by a return close to the average normal level at the end of twenty-four hours. On the other hand, the average fluid content of the white matter and the medulla did not show a significant increase until eight hours after trauma. This increase persisted for twenty-four hours in the cerebral white matter but not in the medulla. The average increases observed in the fluid content were very small, the greatest alteration being an average increase of 1.5 per cent in the cerebral white matter after eight hours. The possible significance of changes of this low order of magnitude will be discussed later.

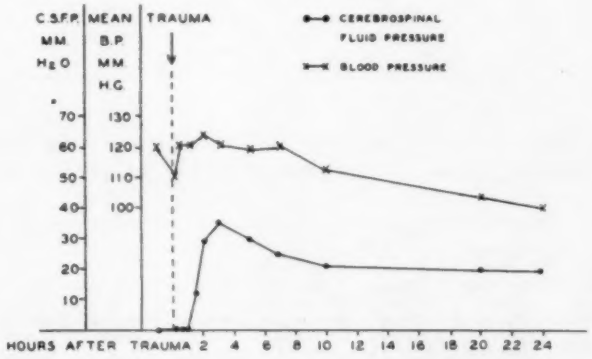

Chart 1 (dog T 56).—Effect of a 500 Gm. weight.

GROUP 3.—*Trauma by a 1,000 Gm. Weight; Animals Killed After Six Hours.*—The animals in group 2 exhibited an inconstant and not marked increase in intracranial pressure. The greatest change in the water content of their brains was found in those killed four or eight hours after trauma. Therefore, in the four dogs of group 3 the degree of trauma was doubled, and the animals were killed six hours after trauma. The results are shown in table 3.

The cerebrospinal fluid pressure was markedly elevated in three animals and moderately elevated in one, and these changes persisted without significant alteration until the termination of the experiments. The mean blood pressure showed some elevation in three of the four animals.

The average fluid contents of the various parts of the brain closely approximated or were slightly lower than the normal values in group 1, except in the cerebral gray matter, where the fluid content was slightly greater than the average normal in three of the four animals.

GROUP 4.—*Trauma by a 1,000 Gm. Weight; Animals Killed at the Time of the Maximum Increase in Cerebrospinal Fluid Pressure.*—Table 4 shows the results in this group of ten dogs. A marked increase in the cerebrospinal fluid

pressure was observed after trauma in eight of the animals (not measured in dog T 17). In the other two, no change occurred. The time of onset and the rapidity of development of the increase of pressure were variable, as shown in chart 2. In most experiments there occurred a sudden rise in pressure of from 30 to 40 mm. immediately after the trauma, followed by a gradual return to the

TABLE 3.—*Fluid Content of the Brains of Dogs Killed Six Hours After Trauma by a 1,000 Gm. Weight*

Dog No.	Immediate Rise in Mean Blood Pressure, Mm. of Hg	Later Rise in Mean Blood Pressure, Mm. of Hg	Cerebrospinal Fluid Pressure: Maximum Increase, Mm. of H_2O	Cerebrospinal Fluid Pressure: Increase at End of Experiment, Mm. of H_2O	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
T11	0	19	65	60	80.4	67.4	79.0	67.6
T12	6	18	90	90	80.5	67.1	80.5	68.5
T13	0	0	20	10	81.2	69.4	80.4	71.9
T15	56	16	85	80	79.2	67.2	79.9	71.2
Average					80.3	67.8	79.9	69.8

TABLE 4.—*Fluid Content of the Brains of Dogs Killed at the Time of the Maximum Rise in Cerebrospinal Fluid Pressure; Trauma by a 1,000 Gm. Weight*

Dog No.	Time Dog Was Killed, Hours After Trauma	Immediate Rise in Mean Blood Pressure, Mm. of Hg	Later Rise in Mean Blood Pressure, Mm. of Hg	Cerebrospinal Fluid Pressure, Maximum Increase, Mm. of H_2O	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
T16	24	31	0	65	80.7	68.5	79.1	71.6
T17	23	44	0	?*	79.6	69.5	80.0	70.4
T18	6½	0	0	95	80.3	68.0	80.2	69.9
T19	24	0	0	±	80.4	68.8	79.6	70.5
T20	3	23	46	125	79.3	68.5	80.6	73.4
T21	5½	92	17	95	80.1	68.5	80.5	71.4
T22	6	4	0	50	78.4	67.9	78.2	71.3
T23	24	0	0	60	80.3	68.5	78.8	71.0
T25	18	4	17	±	82.3	69.4	80.9	71.6
T26	6	95	18	115	78.7	71.4	80.1	66.4
Average					80.0	68.9	79.8	70.7

* Satisfactory readings not obtained due to repeated obstruction of needle.

"pretrauma" level, within about thirty minutes. The subsequent sustained rise usually began from one to two hours after trauma. As indicated in table 4, the period of maximum pressure varied from three to twenty-four hours. Experiments were not conducted longer than twenty-four hours on account of the possible confusing effects of dehydration, recovery from anesthesia and cerebral irritation.

There was no marked change in the mean arterial blood pressure in any of the animals, although some immediate elevation occurred in seven and a later moderate increase over the control level was observed in four.

The values obtained in the determinations of the fluid content were surprisingly constant. In only seven of the forty specimens examined in this group did the fluid content vary more than 1 per cent from the average for the tissue involved. Average values were slightly greater than the normal averages in all tissues except the cerebellar gray matter, which closely approximated the normal level. The greatest average change observed in the fluid content was an increase of 0.9 per cent in the cerebral white matter.

GROUP 5.—*Immediate Effect of the Intravenous Injection of 50 per Cent Solution of Dextrose in Animals Exhibiting Posttraumatic Increase in Cerebrospinal Fluid Pressure.*—In five animals the usual trauma was administered by means of a 1,000 Gm. weight. When a marked, sustained increase in the cerebrospinal fluid pressure had occurred, each animal was given 50 cc. of a 50 per cent solution

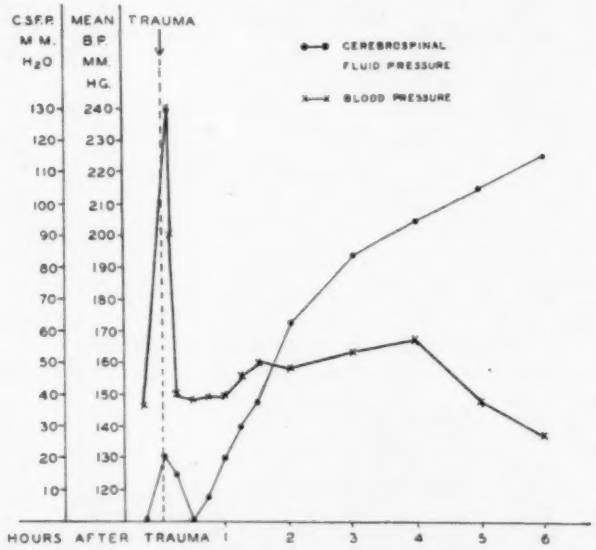

Chart 2 (dog T 26).—Effect of a 1,000 Gm. weight.

of dextrose intravenously. In all instances the cerebrospinal fluid pressure fell to or below the normal level within three minutes after the end of the injection. The animals were killed immediately and their brains were rapidly removed for determination of the fluid content. The results are shown in table 5. The average findings were almost identical with those obtained on the brains of the animals in group 4, to which no dextrose was given (see also table 7), and were, except in the cerebellar gray matter, slightly greater than the average control values.

GROUP 6.—*Effect of the Intravenous Injection of 50 per Cent Solution of Dextrose in Animals Exhibiting Posttraumatic Increase in Cerebrospinal Fluid Pressure and Killed Two Hours After the Injection.*—This group of five dogs was treated as those in group 5 except that they were not killed until two hours after the administration of dextrose. In four of the five animals the cerebrospinal fluid pressure, which had fallen to or below normal after the injection, had again become elevated at the time the experiment was terminated (table 6 and chart 3).

The fluid contents were again almost unexceptionally constant. The average values were not only slightly greater than the control levels but also exceeded slightly the average fluid contents observed in groups 4 and 5. In fact, the values in this group were higher than in any other group except group 2 C (table 7).

TABLE 5.—*Fluid Content of the Brains of Dogs Killed Immediately After Receiving 50 Cc. of 50 per Cent Solution of Dextrose Intravenously; Previous Trauma by a 1,000 Gm. Weight*

Dog No.	Interval Between Trauma and Injection of Dextrose, Hr.	Immediate Rise in Mean Blood Pressure, Mm. of Hg	Later Rise in Mean Blood Pressure, Mm. of Hg	Cerebrospinal Fluid Pressure: Maximum Increase Before Injection, Mm. of H_2O	Cerebrospinal Fluid Pressure: Increase at Time of Killing, Mm. of H_2O	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
T27	3½	20	9	140	±	80.2	69.5	79.6	73.8
T28	3	22	10	130	±	79.6	70.2	79.4	70.8
T29	7½	24	0	80	±	80.0	69.5	81.0	72.3
T30	4	69	0	200	±	80.8	68.9	79.7	72.3
T33	3½	13	0	110	±	79.2	66.6	78.2	80.0
Average						79.9	68.9	79.6	71.8

TABLE 6.—*Fluid Content of the Brains of Dogs Killed Two Hours After Receiving 50 Cc. of 50 per Cent Solution of Dextrose Intravenously; Previous Trauma by a 1,000 Gm. Weight*

Dog No.	Interval Between Trauma and Injection of Dextrose, Hr.	Immediate Rise in Mean Blood Pressure, Mm. of Hg	Later Rise in Mean Blood Pressure, Mm. of Hg	Cerebrospinal Fluid Pressure: Maximum Increase Before Injection, Mm. of H_2O	Cerebrospinal Fluid Pressure: Increase at Time of Killing, Mm. of H_2O	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
T32	7¾	27	0	65	10	81.2	70.4	80.3	72.0
T34	4	0	0	70	50	80.4	69.4	80.4	71.2
T35	3½	3	6	140	80	79.8	68.4	80.5	73.6
T36	5	8	0	40	65	81.0	68.0	80.6	71.7
T37	3	15	0	104	±	79.7	69.1	80.1	70.5
Average						80.4	69.1	80.4	71.8

GROUP 7.—*Lumbar Cerebrospinal Fluid Outflow in Untraumatized Dogs.*—In two dogs the outflow of cerebrospinal fluid from an 18 gage needle (the same needle was used in both experiments and in all experiments in group 8), measured in drops per minute, was determined over a period of twenty-four hours. The needle was inserted into the lumbar subarachnoid space with the animal's head in the holder, as in the preceding experiments, but with the body twisted to lie on its side. The needle was left open at all times.

During the first hour after lumbar puncture the outflow varied greatly from minute to minute, usually with a marked respiratory fluctuation. After this period

of initial variation, the flow gradually assumed a relatively constant rate of one drop in one and one half to two minutes, and this rate was maintained throughout the twenty-four hours of observation.

GROUP 8.—*Effect of Trauma by a 1,000 Gm. Weight on the Lumbar Outflow of Cerebrospinal Fluid.*—The outflow was determined as in group 7, and the same initial fluctuation was observed in all of the six animals. However, a much slower control rate of flow was observed in three of the animals than in the two dogs of group 7. Trauma was administered two hours after lumbar puncture, when the outflow had assumed a constant rate.

The results observed were not consistent or conclusive (table 8). A variable and unsustained increase in flow usually occurred immediately after trauma. At later periods the rate of flow was relatively unchanged in three animals (T 39,

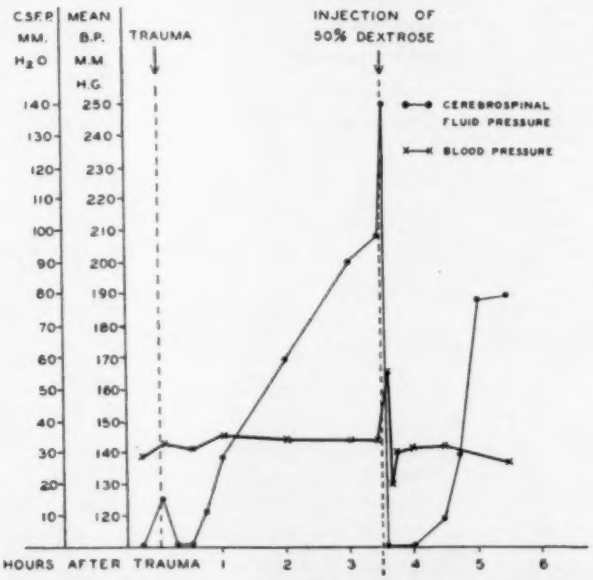
C.S.F.P. MM. H_2O
MEAN B.P. M.M. H.G.
TRAUMA
INJECTION OF 50% DEXTROSE
CEREBROSPINAL FLUID PRESSURE
BLOOD PRESSURE
140 130 120 110 100 90 80 70 60 50 40 30 20 10
250 240 230 220 210 200 190 180 170 160 150 140 130 120
HOURS AFTER TRAUMA 1 2 3 4 5 6

Chart 3 (dog T 35).—Effect of a 1,000 Gm. weight. Fifty cubic centimeters of 50 per cent solution of dextrose was injected three and one-half hours after trauma.

T 40 and T 46), seemed to be definitely slowed in one (T 43) and showed some degree of sustained increase in two (T 42 and T 47).

GROUP 9.—*Effect on Cerebrospinal Fluid Pressure of the Intracisternal Injection of Small Quantities of Physiologic Solution of Sodium Chloride.*—Before evaluating the significance of the small changes observed in the fluid content of the brain, it seemed essential to determine whether such small increases in the fluid content could be responsible for the rise observed in the cerebrospinal fluid pressure. Three dogs were used for this purpose. A three way stopcock was interpolated between the cisternal needle and the manometer, in order that fluid might be withdrawn or injected with only a momentary break in the pressure measurements.

After a normal constant cerebrospinal fluid pressure had been observed for one hour, a volume of physiologic solution of sodium chloride estimated to be equivalent to 0.5 per cent of the weight of the dog's brain was injected into the cisterna magna, and the stopcock was immediately turned back to reestablish communication between the cistern and the manometer. After the cerebrospinal fluid pressure was observed, the stopcock was opened to allow the escape of fluid

TABLE 7.—*Average Fluid Contents of the Brains in the Various Groups of Experiments*

Type of Experiment	Number of Experiments	Fluid in Cerebral Gray Matter, %	Fluid in Cerebral White Matter, %	Fluid in Cerebellar Gray Matter, %	Fluid in Medulla Oblongata, %
1. Normal controls	5	79.4	68.0	79.6	70.9
2a. Brain removed 2 hours after trauma by 500 Gm. weight	5	80.1	68.6	80.6	70.9
2b. Brain removed 4 hours after trauma by 500 Gm. weight	2	80.2	68.5	80.6	70.7
2c. Brain removed 8 hours after trauma by 500 Gm. weight	2	80.4	70.5	81.0	71.0
2d. Brain removed 24 hours after trauma by 500 Gm. weight	3	79.7	69.6	80.4	70.4
2. Average of 2a, 2b, 2c and 2d	11	80.1	69.1	80.6	70.8
3. Brain removed 6 hours after trauma by 1,000 Gm. weight	4	80.3	67.8	79.9	68.9
4. Brain removed at time of maximum cerebrospinal fluid pressure after trauma by 1,000 Gm. weight	10	80.0	68.9	79.8	70.4
5. Brain removed immediately after posttraumatic treatment with 50 per cent solution of dextrose	5	79.9	68.9	79.6	71.8
6. Brain removed 2 hours after posttraumatic treatment with 50 per cent solution of dextrose	5	80.4	69.1	80.4	71.8

TABLE 8.—*Outflow of Cerebrospinal Fluid Through a Lumbar Puncture Needle After Trauma by a 1,000 Gm. Weight*

Dog No.	Initial Rate, Drops per Minute	Immediately After Trauma, Drops per Minute	1 Hour After Trauma, Drops per Minute	2 Hours After Trauma, Drops per Minute	4 Hours After Trauma, Drops per Minute	24 Hours After Trauma, Drops per Minute
T39	1	3	1	1	1	3/4
T40	1/2	2	1	1/2	1/2	1/3
T42	1/6	11	1/3	1/2	1	—
T43	1/7	3/4	1/5	1/5	1/25	0
T46	1/2	2	1/2	1/3	0	—
T47	1/5	1/5	1/3	1/2	3/4	0

until the pressure had been reduced to the normal level. The entire procedure was then repeated. In this way, several readings were obtained on each animal. Subsequently, twice the volume of saline solution originally employed was injected and pressure readings made as before, and finally the volume was again doubled to obtain another series of readings. These approximate volumes were employed because most of the average increases observed in the fluid content of the brain after trauma did not exceed 1 per cent. After the readings were completed, the brain was removed, free fluid blotted away and the brain weighed.

The results are shown in table 9. Only immediate pressure readings are included, since it was the purpose of these experiments to determine only the

effects of the actual increases in the volume of the intracranial contents brought about by the injections. After injection of an amount of fluid equivalent to approximately 0.5 per cent of the weight of the brain, a definite but relatively slight rise in the cerebrospinal fluid pressure was observed, the greatest increase being 15 mm.

When a volume equivalent to approximately 1 per cent of the weight of the brain was injected, a greater increase in pressure was observed, the greatest being 60 mm. and the average 31 mm. This degree of rise in pressure is comparable to that found in some of the animals subjected to mild trauma (table 2) but does not approach that usually observed after more severe trauma (tables 3, 4, 5 and 6).

TABLE 9.—*Effect on Cerebrospinal Fluid Pressure of the Intracisternal Injection of Small Volumes of Physiologic Solution of Sodium Chloride*

Dog No.	Body Weight, Kg.	Weight of Brain, Gm.	Volume of Saline Solution Injected, Cc.	Percentage of Weight of Brain of Saline Solution Injected	Immediate Increase in Cerebrospinal Fluid Pressure, Mm. of H_2O	Increase in Cerebrospinal Fluid Pressure After 15 Sec.
T58	11.47	76	0.35	0.46	20	15
			0.35	0.46	15	10
			0.70	0.92	60	50
			0.70	0.92	55	40
			1.40	1.84	390	260
			1.40	1.84	450	280
T59	12.0	73	0.35	0.48	10	5
			0.35	0.48	5	±
			0.70	0.96	20	15
			0.70	0.96	10	5
			0.70	0.96	10	±
			1.40	1.92	120	100
			1.40	1.92	160	140
			0.70	0.96	15	10
T60	10.4	62	0.3	0.48	5	±
			0.3	0.48	10	5
			0.6	0.97	25	15
			0.6	0.97	40	25
			0.6	0.97	30	20
			1.2	1.94	205	150
			1.2	1.94	165	120

Injection of amounts of fluid equivalent to approximately 2 per cent of the weight of the brain produced an immediate marked increase in the cerebrospinal fluid pressure. This degree of increase was greater than that usually observed after severe trauma.

COMMENT

Before arriving at conclusions from the experiments reported herein, it is necessary to consider the several essential limitations inherent in the method of study.

It is impossible to determine the fluid content of the brain both before and after trauma in the same animal. Therefore, the only available basis for comparison consists of average findings in normal animals. This objection is obviated to some extent by the small degree of variation observed in the normal animals.

Similarly, in the experimental animals, in spite of occasional individual variations, the great majority of the individual findings did not vary significantly from the average of their groups.

After careful study of the tables, it seems justifiable to conclude that any change in average findings as great as 1 per cent of fluid content may be considered as beyond the probable limits of error of the method.

If the validity of the results is accepted within these limits, it is next in importance to recognize what degree of increase in the total intracranial contents is necessary to produce a significant increase in the cerebrospinal fluid pressure. The results of the experiments on the animals in group 9 (table 9) indicate a marked increase in the degree of rise in pressure resulting from increasing the intracranial contents more than the equivalent of 1 per cent of the weight of the brain. In other words, 1 per cent of the weight of the brain may be considered as the limit beyond which the quantity of intracranial contents cannot be increased without producing a marked rise in pressure. It is apparent that an increase in the tissue fluid comparable to that found in edematous tissues elsewhere in the body (from 2 to 6 per cent in tissue as dense as muscle [24]) could exist in the brain only if the remaining intracranial contents (i. e., blood and cerebrospinal fluid) were greatly diminished.

The rise in cerebrospinal fluid pressure observed in these experiments could conceivably arise from an increase in tissue fluids, in cerebrospinal fluid or in intracranial blood volume or from any combinations of alterations in their respective volumes which resulted in an increase in the total intracranial fluid volume.

A survey of the composite average results shown in table 7 shows at once that no great change in tissue fluids has resulted from trauma to the head in these experiments. In only two groups (2 and 6) were as many as two of the four types of brain tissue examined found to have as much as 1 per cent greater fluid content than was found in the average normal animal. On account of the striking uniformity of the results, it may be justifiable to conclude that a small increase in fluid content (particularly of the gray matter) probably follows trauma to the head. But the degree of increase observed

24. Pilcher, C.; Calhoun, J. A.; Cullen, G. E., and Harrison, T. R.: Studies in Congestive Heart Failure: V. The Potassium Content of Skeletal Muscle Obtained by Biopsy, J. Clin. Investigation **9**:191, 1930. Harrison, T. R.; Pilcher, C., and Ewing, G.: Studies in Congestive Heart Failure: IV. The Potassium Content of Skeletal and Cardiac Muscle, ibid. **8**:325, 1930.

was not in proportion to and probably should not be considered as the sole cause of the increase in cerebrospinal fluid pressure which occurred.

The results of the experiments on the cerebrospinal fluid outflow, as stated previously, were not consistent or conclusive (table 8). From them it may be said only that the outflow of cerebrospinal fluid is increased in volume in some cases after trauma to the head, but this may not necessarily be an index of the production of cerebrospinal fluid, since the outflow would probably be increased by any alteration in intracranial contents producing increased pressure. Of interest in this connection are the findings of Hoff,[20] who injected fluorescein intravenously in estimating the production of cerebrospinal fluid and found an increase after measured trauma to the head. Recent experiments by Parker and Lehman [25] have demonstrated that a rise in the cerebrospinal fluid pressure occurs after replacement of small amounts of cerebrospinal fluid with equal volumes of blood serum, defibrinated blood or solution of hemoglobin but not after replacement with washed red blood cells. The authors concluded: "It is probable that the phenomena reported are the result of the increase of osmotic pressure of the cerebrospinal fluid due to the introduction of blood proteins."

The results of experiments on the effect of hypertonic solution of dextrose (table 5 and 6) confirm, for the first time in traumatized animals, the original observations of Weed and McKibben,[26] Weed and Hughson [27] and many later investigators on the reduction of cerebrospinal fluid pressure after the intravenous administration of these solutions. Further, they are not incompatible with the alterations in the bulk of the brain found by Weed and McKibben [28] after intravenous injections of solutions of various concentrations, for these alterations may well have been due to changes in such factors as ventricular size or intracerebral blood volume.

The results of the experiments reported herein also confirm the findings of Masserman,[29] who measured the cerebrospinal fluid pressure

25. Parker, W. P., and Lehman, E. P.: Personal communication to the author.

26. Weed, L. H., and McKibben, P. S.: Pressure Changes in the Cerebro-Spinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, Am. J. Physiol. **48**:512, 1919.

27. Weed, L. H., and Hughson, W.: Systemic Effects of the Intravenous Injection of Solutions of Various Concentrations, with Especial Reference to the Cerebrospinal Fluid, Am. J. Physiol. **58**:53, 1921.

28. Weed, L. H., and McKibben, P. S.: Experimental Alteration of Brain Bulk, Am. J. Physiol. **48**:531, 1919.

29. Masserman, J. H.: Effects of Intravenous Administration of Hypertonic Solutions of Dextrose, with Special Reference to the Cerebrospinal Fluid Pressure, J. A. M. A. **102**:2084 (June 23) 1934.

in untraumatized human subjects receiving intravenous injections of hypertonic solutions. He observed an original fall in pressure followed by a rise to a level equal to or greater than the control value. The present experiments suggest that in traumatized dogs concomitant with this secondary rise in pressure, there may be an increase in the cerebral tissue fluid greater than that present before the injection. These findings cast considerable doubt on the wisdom of the intravenous use of hypertonic solutions in the treatment of injuries of the head.

Regarding intracranial blood volume, there is little evidence. Wolff and Blumgart [30] found a decrease in the velocity of blood flow through the brain after increasing the cerebrospinal fluid pressure through a needle in the cisterna magna. However, they believed that "through cerebral vasodilatation, the flow, though slowed, is increased in volume . . . ." Pilcher,[31] using the arteriovenous oxygen difference method, found a decrease in the volume flow of blood through the brain after the intravenous injection of hypertonic solutions in normal dogs. The only observations on the actual intracerebral blood volume are those of Weil, Zeiss and Cleveland,[32] who injected colloidal silver intravenously and determined the silver content of the brain (which does not remove colloidal silver from the blood). They found an increase in the blood volume of the brain immediately (from five to fifteen minutes) after the intravenous injection of 15 per cent sodium chloride. There are no recorded observations pertaining to intracranial blood volume after cerebral trauma. It seems probable that this may be an important factor in the mechanism producing the increased cerebrospinal fluid pressure which follows trauma to the head.

SUMMARY AND CONCLUSIONS

The fluid content of various portions of the dog's brain has been determined after measured trauma of varying degrees.

After trauma sufficient to produce a definite elevation in the cerebrospinal fluid pressure, the fluid content of the brain was slightly, if at all, increased above the average normal level. The small average

30. Wolff, H. G., and Blumgart, H. L.: The Cerebral Circulation: VI. The Effect of Normal and Increased Intracranial Cerebrospinal Fluid Pressure on the Velocity of Intracranial Blood Flow, Arch. Neurol. & Psychiat. **21**:795 (April) 1929.

31. Pilcher, C.: Cerebral Blood Flow: I. The Effect of Intravenous Administration of Hypertonic and Hypotonic Solutions on the Volume Flow of Blood Through the Brain, Arch. Neurol. & Psychiat. **24**:899 (Nov.) 1930.

32. Weil, A.; Zeiss, F. R., and Cleveland, D. A.: The Determination of the Amount of Blood in the Central Nervous System After Injection of Hypertonic Solutions, Am. J. Physiol. **98**:363, 1931.

increase observed is not believed to be sufficiently great to account for the elevation of the cerebrospinal fluid pressure.

There is no conclusive evidence in these studies, or in those of other authors, indicating the existence of marked posttraumatic cerebral edema.

The outflow of lumbar cerebrospinal fluid after trauma to the head was not consistently altered but in some instances showed a sustained increase.

It seems probable that other factors, such as cerebrospinal fluid volume and intracranial blood volume, are of greater importance than cerebral edema in producing the increased intracranial pressure which follows trauma to the head.

DISLOCATION AND FRACTURE-DISLOCATION OF LOWER CERVICAL VERTEBRAE

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Nearly as far back as one is able to peruse the annals of medical history one finds references to fractures and fracture-dislocations of the cervical portion of the spine. An injury involving this segment of the spine rightly struck terror into the heart of the surgeon who was called on to treat it. About 2500 B. C. the timidity of a surgeon regarding such an injury was recorded in a papyrus written during that period, in which it was stated:[1] "One having a crushed vertebra in his neck, he is unconscious of his two arms and his two legs, and he is speechless. An ailment not to be treated." The reluctance to treat such a lesion can only be surmised.

Hippocrates[2] formed a rational and sound plan for the treatment of a lesion of this type in the acute stage. His method consisted in extension of the head with the patient in a recumbent position. Despite his teachings his contemporaries were prone to practice a form of therapy called succussion. The unfortunate patient was placed on a vertical ladder with the head down. The ladder was then shaken forcefully in the hope that reposition of the involved vertebra might take place. Needless to say, the torture experienced by the patient was terrific, and the mortality rate was high. The father of medicine decried such a brutal manner of treatment but his laments were of no avail at that time. It is to him, however, that the modern nonoperative form of treatment for such a lesion owes its origin, although various physicians have introduced minor modifications of his principle.

Guy de Chauliac[3] and others who dominated the surgical world about the fifth century used much the same method of treatment as

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1. Breasted, James H.: The Edwin Smith Surgical Papyrus, Chicago, University of Chicago Press, 1930.

2. The Genuine Works of Hippocrates, translated by Francis Adams, New York, William Wood & Company, 1929.

3. de Chauliac, Guy: On Wounds and Fractures, translated by W. A. Brennan, Chicago, W. A. Brennan, 1923.

Hippocrates. Paré,[4] who contributed greatly to the knowledge of traumatic lesions, obtained many good results by traction exerted on the extended head.

Skepticism as to the wisdom of attempting reduction of a fracture of a cervical vertebra was universal during the early part of the nineteenth century. Boyer[5] said: "Any attempt at setting these fractures would not only be useless but dangerous, by the straining it would occasion." Cupping and scarification of the cervical region, as well as the use of setons and ointments, were recommended by him, and these appear to have been the only armamentarium resorted to by the surgeons of his day.

Chelius[6] admitted that the patient with such a fracture might be left alone. He said that the pain would gradually subside and that the patient would have no inconvenience other than an unnatural position of the head and restricted motion. Fearful lest the spinal cord be torn asunder by manipulation or matters be made worse, Dupuytren,[7] and others agreed to this policy of laissez faire. The attitude of the medical profession in regard to lesions of this nature can be summed up in the words of Abernethy:[8] "They are cases so generally fatal, that I am convinced an assembly of old surgeons would doubt that there was a possibility of recovery in any case." One is thankful that these teachings as regards traumatic lesions of the cervical portion of the spine in the acute stages have been discarded.

In 1814 Henry Cline performed what is now known as laminectomy for the removal of pressing vertebral arches. His operation was designed primarily for the treatment of fracture of the spine, but at that time the operation was met with a storm of criticism. A strong controversy arose between Sir Astley Cooper[9] and Sir Charles Bell as to the value and propriety of resection of the vertebral arches. As a result of this operation, as well as of the controversy which arose, the medical profession was shaken from the doldrums of thought in regard to these lesions, and greater interest was stimulated.

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4. Paré, Ambroise: The Workes of that Famous Chirurgion Ambrose Parey, translated by T. Johnson, London, Mary Clark, 1678.

5. Boyer, Alexis: The Lectures of Boyer upon Diseases of the Bones, arranged by A. Richerand and translated by M. Farrell, London, John Callow, 1807.

6. von Chelius, M. J.: System of Surgery, translated by John F. South, London, Henry Renshaw, 1845.

7. Dupuytern, Guillaume: On the Injuries and Diseases of Bones, translated and edited by F. leGros Clark, London, Sydenham Society, 1847.

8. Abernethy, John: Lectures on Anatomy, Surgery and Pathology, London, James Bullock, 1828.

9. Cooper, Astley, P.: A Treatise on Dislocations and on Fractures of the Joints, ed. 2, London, Longman, Hurst, Rees, Orme and Browne, 1823.

Ashhurst [10] in 1867 reviewed the literature on the subject. The treatise which he wrote is an admirable one, constituting a profound advancement in the knowledge of this type of lesion in regard to diagnosis and the pathologic picture. Concerning paralysis, he said:

> The cause of paralysis in some cases is obscure. In fatal cases the post-mortem appearances have consisted in effusion of blood with softening of the cord structure, but in some cases which have recovered, the paralysis has been so ephemeral as to give rise to the impression that the cause has been congestion rather than actual extravasation.

In regard to diagnosis, he stated:

> If paralysis below the seat of injury be immediate, complete and permanent, there is every reason to fear that the cord has been absolutely divided. If paralysis is immediate but not permanent it is probably a case of contusion of the cord without structural damage. If paralysis comes on as a late sign, increases to a certain point, then to diminish again, it is probably an effusion, within or without the cord which, under favorable circumstances, may be absorbed. If paralysis is progressive, if not arrested, it will invariably sooner or later prove fatal.

From the literature Ashhurst collected reports of 212 cases of injury to the cervical region of the spine. The mortality rate in this series was 77 per cent; 18 per cent of the patients recovered from the injury and 3 per cent were relieved. The treatment was by extension, resection or simple general care of the patient.

In 1869 Blasius [11] collected reports of 159 cases of definitely diagnosed dislocation of the cervical portion of the spine. In 123 of these cases the injury proved fatal, while in 36 there was improvement. In the cases of bilateral forward dislocation, the severest type, there was a mortality rate of 92.6 per cent.

These startling statistics alarmed and aroused surgeons who saw the inadequacy of the treatment for this type of lesion. A method of reduction was described by Walton [12] in 1889. This method is still in use at the present time and is called the retrolateral flexion maneuver with rotation. Walton reported 5 cases of dislocation of the cervical vertebrae in which he used this treatment. A few years later Wagner and Stolper [13] described a method of reduction which, in the main, is similar to that of Walton.

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10. Ashhurst, John, Jr.: Injuries of the Spine, Phliadelphia, J. B. Lippincott & Co., 1867.

11. Blasius, E.: Die traumatischen Wirbelverrenkungen, Vrtljschr. f. d. prakt. Heilk. **102**:1, 1869; **103**:46, 1869; **104**:77, 1869.

12. Walton, George L.: Dislocation of the Cervical Vertebrae, Boston M. & S. J. **120**:277, 1889.

13. Wagner, W., and Stolper, P.: Die Verletzungen der Wirbelsäule und des Rückenmarks, in Billroth, T., and Luecke, G. A.: Deutsche Chirurgie, Stuttgart, F. Enke, 1898, no. 40, p. 385.

With the advent of the roentgen rays, one became able to evaluate each lesion. This led to a safer and saner manner of treatment and improved results.

Taylor [14] in 1929 described a method of reduction by means of forceful traction exerted on the extended head. Nine persons were treated by him. After reduction had taken place, the head and neck were immobilized in a plaster helmet. He stated that immobilization was imperative until bony ankylosis had taken place between the vertebral bodies and that bony ankylosis always took place.

One year later Langworthy [15] reported 30 cases of dislocation of the cervical vertebra in which treatment was given by him. Reduction was obtained by traction in cases of unilateral as well as of bilateral injury. The mortality rate in this series was 30 per cent. Diagnosis of a unilateral dislocation was made from physical examination and by the fact that the patient was cured by manipulation of the head.

The literature on fracture-dislocation of the cervical portion of the spine is voluminous; yet nearly all of the articles concern the lesion in the acute stage. Many persons with injury to the cervical portion of the spine survive the acute stage but have residual symptoms. Reports of series of cases which have been cited deal mostly with the condition in the acute stage.

Only a limited number of articles could be found which dealt with the residual effects of the more acute lesions and their treatment. De Quervain and Hoessly [16] in 1917 transplanted the spine of the scapula between adjacent cervical vertebrae to produce a fusion of these in an attempt to relieve 2 patients suffering from the residual symptoms caused by an earlier fracture-dislocation. As far as the function of the neck and the relief of preoperative symptoms were concerned, the results were reported as excellent. Hibbs [17] in 1922 reported a case of fracture-dislocation of the cervical portion of the spine in which fusion was employed to relieve the patient from symptoms arising from a previously treated injury of that portion of the spine. The patient was greatly benefited by this procedure. In 1930 Krida [18] reported a dislocation of the fifth cervical vertebra in a patient on whom repeated attempts at

14. Taylor, Alfred: Fracture Dislocations of the Cervical Spine, Ann Surg. **90**:321, 1929.

15. Langworthy, M.: Dislocations of the Cervical Vertebrae, J. A. M. A. **94**: 86 (Jan. 11) 1930.

16. de Quervain, F., and Hoessly, H.: Operative Immobilization of the Spine, Surg., Gynec. & Obst. **24**:428, 1917.

17. Hibbs, Russell A.: Fracture-Dislocation of the Spine Treated by Fusion, Arch. Surg. **4**:598 (May) 1922.

18. Krida, Arthur: Dislocation Between Fifth and Sixth Cervical Vertebrae: Delayed Operative Reduction and Fusion, Am. J. Surg. **9**:560, 1930.

closed reduction had been carried out, all of which were followed by redislocation. After these attempts a portion of the cervical segment of the spine was fused; the patient was relieved from symptoms as well as from the danger of redislocation.

Cases in which the patient presents the residual effects of trauma to the cervical region have been little discussed, and it is the purpose of this paper to evaluate spinal fusion and laminectomy in reference to treatment and prognosis in cases in this category.

PRESENTATION OF CASES

Between 1923 and 1933 18 patients with dislocation or fracture-dislocation of the lower cervical vertebrae were admitted to the New

TABLE 1.—*Diagnosis and Type of Operation in Series of Cases* *

Case	Diagnosis	Operation
1	Fr.-dis. 5 C	Spinal fusion 4 C-7 C; laminectomy 4 C and 5 C
2	Fr.-dis. 3 C	Spinal fusion 2 C-7 C
3	Fr. 6 C	Spinal fusion 5 C-7 C
4	Fr. 5 C	Spinal fusion 4 C-6 C
5	Fr.-dis. 5 C	Spinal fusion 4 C-6 C
6	Dis. 6 C	Spinal fusion 6 C-1 D
7	Fr.-dis. 6 C	Spinal fusion 5 C-1 D
8	Dis. 4 C	Spinal fusion 2 C-5 C
9	Fr.-dis. 5 C	Spinal fusion 4 C-6 C
10	Dis. 5 C	Spinal fusion 3 C-6 C; laminectomy 3 C-6 C
11	Fr.-dis. 4 C	Spinal fusion 3 C-6 C; laminectomy 4 C-5 C
12	Fr.-dis. 5 C	Spinal fusion 5 C-7 C
13	Fr. dis. 6 C	Spinal fusion 5 C-7 C
14	Dis. 4 C	Spinal fusion 4 C-5 C; laminectomy 4 C
15	Dis. 5 C	Spinal fusion 4 C-6 C
16	Dis. 5 C	Spinal fusion 5 C-7 C
17	Fr.-dis. 6 C	Spinal fusion 5 C-7 C; laminectomy 5 C
18	Fr.-dis. 4 C	1. Spinal fusion 3 C-6 C 2. Laminectomy 4 C-5 C 3. Laminectomy 3 C-6 C; partial excision of vertebral body 5 C

* The abbreviations may be interpreted as follows: Fr.-dis. 5 C, fracture-dislocation of the fifth cervical vertebra, etc.

York Orthopaedic Dispensary and Hospital. The average age of these patients was 29, the youngest being 19 and the oldest 55. Fourteen of the patients were men, and 4 were women. With the exception of 2 patients, the original trauma had taken place some time prior to the patient's first visit to this hospital. Two patients were seen two days after the accidents which were responsible for the lesions, while 1 was seen two weeks after the original trauma. The longest interval was sixteen years, and the average interval, sixteen months. Although there are 2 patients in the series who could be designated as having fracture-dislocation of the more acute type, the entire series is regarded as presenting the residual manifestations of acute traumatism of the lower cervical segment of the spine.

The diagnosis and type of operation performed in each case are recorded in table 1.

CAUSE OF FRACTURES AND FRACTURE-DISLOCATIONS OF THE CERVICAL PORTION OF THE SPINE

The cervical portion of the spine has a wide range of mobility, which is dependent on many factors. The ligaments are long enough to allow great mobility, and they do not check flexion before the anterior lips of the bodies are about in contact. The ligamentum nuchae, which in the pronograde mammals acts as a powerful check to flexion, is almost useless for this purpose in man because it is not so fully developed. The articular capsules of the lateral articulations are thin and redundant in the cervical portion of the spine. The capsules, by their inability to constrain the articulations from excessive motion, permit them to glide forward into a luxated state. The cervical bodies are small, separated from each other by a relatively greater amount of intervertebral substance than is found in other areas of the spine. Likewise the lateral articulations are on a more horizontal plane, not only facilitating the motion of flexion but increasing the ease of dislocation.

Mechanically the cervicodorsal junction is unstable. It is a point where the rather flexible cervical portion of the spine meets the comparatively immobile dorsal segment. Naturally, a force exerted on the flexible segment, if of sufficient strength, will cause damage to it rather than to the relatively immobile portion. Forces tend to be concentrated on the areas where fixed and movable portions of the spine meet, as at the cervicodorsal junction.

The cervical segment of the spine, being deficient in so many stabilizing factors, having a mechanical situation which renders it vulnerable and yet being called on to furnish great ranges of mobility, gains its stability at the expense of voluntary and involuntary muscular effort. By involuntary muscular effort is meant the neuromuscular mechanism which maintains the head and neck in an erect posture during the conscious state. The muscles surrounding the cervical portion of the spine are many, and collectively they are very strong. Acting under voluntary control against a given force, these muscles are capable of protecting the spine in most instances. There are times, however, when this muscular control is off guard or is not of sufficient strength, and it is during these unguarded moments that injury to the cervical segment of the spine takes place. The degree of injury which may ensue depends on the extent of the force applied, as well as on the duration of the unguarded moment.

In this series of cases it was almost uniformly found that injury to the cervical portion of the spine had been received during a moment when the spine was not protected by a sufficient amount of muscular effort, as well as when a force producing acute flexion was applied. Seven of the 18 patients gave a history of having struck the bottom

of a swimming pool while diving. As the occiput struck the unyielding bottom the oncoming weight of the trunk and legs forced the head into acute flexion. Two patients were playing football and, while in a sitting position, were struck on the back of the head by another player, thus forcing the head into acute flexion. Injuries had been received in 2 instances when patients had fallen down stairs, bending the head forward during the fall. One case was unusual in that the injury had been received when the patient fell from the height of a few feet, landing on the buttocks. Perhaps in this case injury to the cervical portion of the spine had been caused by the snapping forward of the head, an accident which sometimes occurs in such a fall. The exact manner of the injury was not stated by a few patients.

The series, although small, emphasizes that the mechanism which causes fractures and dislocations in this area of the spine is flexion.

ORIGINAL TREATMENT

The original treatment in 3 of the 18 cases was rest in bed for a few weeks. Reduction of the lesion was attempted in 4 instances by the use of traction on the head. In 1 case traction on the head was followed by the application of a head helmet. This helmet was worn for a few weeks after its application. Manipulation of the head, followed by no supportive apparatus, was the original treatment in 3 cases. In 2 cases osteopathic or chiropractic manipulations were resorted to by the patients. Three patients were not seen elsewhere before they came to this hospital, while 2 did not present any history as regards original treatment.

PREOPERATIVE STATUS OF PATIENTS

Disability was the reason for each of the patients in this series applying to the hospital for treatment. Disability from this type of lesion, whether it is in an acute or a chronic stage, is dependent chiefly on the type and extent of injury to the cord, but partly on the degree of involvement of nerve roots and partly on the extent and type of structural damage to the vertebral column with its supportive structures.

Injury to the cord at the time of primary trauma can occur as the result of concussion, contusion, edema, hematomyelia or laceration. The degree of damage to the cord with its resultant syndromes depends on the type, extent and location of any one or a combination of these. After the integrity of the cord has been affected in any of the foregoing manners, its function may return gradually and, in the milder lesions, will sometimes return to normal, the patient suffering no disability because of the lesion of the cord. As a result of irreparable damage to the cord at the primary trauma, the disability of the patient may be of any degree and of a lasting nature, despite perfect anatomic

reduction. Some of the factors which may operate in causing continued damage to the cord are abnormally luxated vertebrae, pressing vertebral arches and bodies and sclerosis of the coverings of the cord.

To diagnose the presence of involvement of the cord is usually easy. This was true in most of these cases, yet it was difficult to form a satisfactory opinion as to the nature of the pathologic process causing the dysfunction. In the presence of dysfunction of the cord it is sometimes difficult to ascertain whether the dysfunction is due to the original trauma or to latent factors.

The neurologic syndromes, indicating the evidence of involvement of the cord in these cases, will not be presented in detail, but it may be stated that 3 patients with severe syndromes were bedridden when they were first seen at this hospital. An atypical Brown-Séquard phenomenon was present in 1 of these, the lesion being at the level of the fifth cervical segment. Milder and moderate syndromes included atrophy, hemiplegia and contracture as well as other evidence of damage to the cord.

A fracture-dislocation in the cervical portion of the spine, perfectly reduced, may still produce disability in the function of the neck. Such disability may be caused by mobility at fractures of the posterior elements, arthritic productions arising from traumatized lateral articulations, scar formation from the tearing of ligaments or articular capsules and unstable vertebrae.

If unreduced, a malalined cervical segment of the spine may also produce symptoms referable to the head and neck. These symptoms may occur as a result of the aforementioned causes, as well as from impingement of the spinous processes and laminae.

Eight patients in the series had partial rigidity of the neck associated with severe pain in the cervical region. In another instance there was severe pain in this region without any limitation of the motion of the head. Limited motion and an unnatural position of the head were complained of by 1 patient. Stiff neck, limited motion of the head of a mild degree, a grating sensation of the cervical region and a moderate ache in the posterior cervical region were complained of by many patients. All of these symptoms were indicative of a pathologic process of the ligamentous and bony structure of the spine.

Many patients in the series complained of symptoms which would indicate involvement of the nerve roots. In cases of injury in which the cervical segments of the cord were damaged it was difficult in some instances to ascertain whether there was sufficient evidence to warrant the diagnosis of the presence of a pathologic process involving the nerve roots. The radicular type of pain, various atrophies, contractures or sensory disturbances involving the upper extremity would lead one to

suspect trauma to the cervical nerve roots in many patients. Some of the patients presented no evidence of involvement of the cord, and yet there were neurologic syndromes present which, without doubt, could be explained only by the presence of a radicular lesion.

Patients with evidence of involvement of the nerve roots suffered neuralgic pains down the arms and weakness and atrophy of the various muscle groups of the scapula and humerus and of the arm, forearm and hand. Hypesthesias and paresthesias were also frequently present.

A syndrome referable to the head and neck, which was caused by either a bony or a ligamentous pathologic process, has been designated as a vertebral syndrome. The degree of the syndromes arising from involvement of the vertebral column, the nerve root and the spinal cord is given in table 2. In practically all of the cases there was a combination of one or more of these syndromes in the preoperative status of each case.

TABLE 2.—*Preoperative Status of Patients*

Case	Vertebral Syndrome	Nerve Root Syndrome	Cord Syndrome
1	None	Moderate	Severe
2	Moderate	None	None
3	Mild	Moderate	None
4	Mild	Mild	Moderate
5	Mild	Moderate	None
6	Moderate	None	None
7	Mild	Moderate	Mild
8	Moderate	Mild	None
9	Moderate	Mild	None
10	Moderate	Mild	None
11	Moderate	Mild	Severe
12	Moderate	None	Mild
13	Moderate	None	None
14	Mild	Moderate	None
15	Moderate	Mild	None
16	Mild	Moderate	None
17	Mild	Mild	Severe
18	Moderate	Moderate	Mild

PREOPERATIVE ROENTGENOGRAMS

Since there were only 10 cases in which preoperative and postoperative roentgenograms were available, only these will be referred to in connection with roentgenography. As a rule, anteroposterior and lateral views are taken of this type of lesion; however, stereoscopic and oblique roentgenograms are made in special instances. Special views are seldom necessary because these rarely give information in regard to the lesion which cannot be estimated from the routine views.

Anterior displacement was present in all of these cases, the amount of displacement of the luxated vertebrae varying from 5 to 80 per cent of the total horizontal diameter of the vertebral body. The amount of anterior vertebral tilt ranged from a minimum of 12 degrees to a maximum of 58 degrees, and tilt was present in all of the cases. Wedging constitutes a decrease in the vertical diameter of the involved

vertebra anteriorly, while compression constitutes a decrease in the vertical diameter both anteriorly and posteriorly. In many of the cases there was a combination of these. In 5 cases there was no wedging, while in the remaining 5 cases this varied from 6 to 17 per cent of the anterior vertical diameter of the involved vertebra. Compression was not present in 6 cases, and in 4 it ranged from a few to 20 per cent. A lateral wedging of 7 per cent was a feature in 1 case, while in the remaining cases this deformity was not present. Bony ankylosis was not found in any of the 10 cases studied roentgenographically. This is of interest in that the patients were seen many months and sometimes years after their primary injury had occurred.

In case 2 (fig. 1) the body of the fifth cervical vertebra was fractured vertically, with the anterior fragment displaced anteriorly 15 per cent and the posterior fragment displaced posteriorly 30 per cent. In case 9 (fig. 2) there was a fracture-dislocation of the fifth cervical vertebra, but the spinous processes of the fourth and the fifth cervical vertebra were widely separated. Separation should have taken place between the spines of the fifth and sixth cervical vertebrae, unless a fracture of the posterior elements was present. The roentgenographic appearance was indicative of a fracture of the posterior elements, and in this instance operative findings confirmed the diagnosis. Arthritic deposits were a feature in 1 instance and could well account for the complaints referable to the vertebral column. An ununited fracture of the arch could be seen in only 1 case, while in another instance there was the appearance previously noted which suggested a fracture.

In all of the cases in the series diagnosis as to the nature of the lesion was made by roentgenographic examination.

TREATMENT

In 3 cases in which the patient was seen fairly recently after injury an attempt was made to reduce the lesion. This was done by means of 10 pounds of traction on the head exerted with the head in a slightly extended position. Complete reposition of the involved vertebra was not accomplished in any instance, yet relief from pain was experienced by the patient, and the muscular spasm was diminished. Forceful extension was not used to reduce the lesion in any instance. Preoperative care consisted mainly in the partial immobilization of the spine by sand-bags placed on either side of the patient's head, while pain was relieved by rest and the use of sedatives. The patients were operated on within a few days after entering the hospital.

In 13 cases fusion of the spine was carried out. A combined one stage laminectomy and spinal fusion were performed in 5 other instances. When spinal fusion alone was done, the average operating time was one hour and twenty-five minutes; the longest operating time was one

Fig. 1 (case 2).—Roentgenogram showing severe compression fracture of the body of the fifth cervical vertebra.

Fig. 2 (case 9).—Roentgenogram showing a fracture-dislocation of the fifth cervical vertebra.

hour and fifty minutes, while the shortest time was forty-five minutes. In those cases in which the combined procedures were done the average operating time was one hour and fifty minutes. Two hours and thirty-five minutes was the maximum operating time, while the minimum was one hour and twenty minutes.

In 1 instance the operator attempted to replace the dislocated articular facets without success. In another instance a freely movable fractured arch was encountered. This was removed lest the cord be damaged at a later date. In 1 case further surgical work was done. In only 1 case were the coverings of the cord opened intentionally at the primary operation.

Fig. 3 (case 18).—Preoperative roentgenogram showing the greatest degree of anterior vertebral tilt and the greatest percentage of anterior displacement of the involved vertebra observed in any case in the series.

The Hibbs type of spinal fusion was carried out in all instances. This operation consists in the subperiosteal exposure of the laminae, spinous processes and lateral articulations of the vertebrae to be fused. The ligamentum flavum and cartilage from the lateral articulations were removed. Lateral articulations and their processes were then comminuted. Interdigitating bone chips taken from the spinous processes and laminae were turned in such a manner as to bridge across the intervening interlaminar spaces. In the 5 cases in which laminectomy was performed, a bilateral type of laminectomy was done, followed by fusion of the portions of the laminae which were not removed, as well as of the lateral articular processes.

During the operation the patient was in a prone position on the operating table, with the head supported in a head rest. The head rest consisted of a tripod with a horizontal padded loop, which fitted the conformity of the face. With this type of head rest the anesthetist was not hampered in his work, the patient was in a comfortable position, and his head could be changed at any angle desired by the operator.

Because of the proximity of vital centers, surgical intervention in this area of the spine is dangerous unless caution is practiced by the operator. The cervical segment of the spine lies rather deep, so that an extensive exposure is necessary. Usually exposure of the spinous processes of two vertebrae above and two below the seat of injury is required, as this will insure a sufficient operative field with the minimum of retraction force. When spinal fusion or laminectomy of this area of the spine is performed, it should be borne in mind that the posterior elements are smaller than elsewhere and the interlaminar spaces are wider. Needless to say, curetting of ligaments, turning down of bone chips or removal of the laminae should be done with the utmost care.

Immediately after the operation the usual care was the application of a few pounds of traction on the head with the head immobilized by sand-bags. In a few instances sand-bags alone were used, but traction on the head not only aids in immobilization but tends to relieve muscular spasm. The head is placed in a slightly extended position by means of a small pillow beneath the patient's shoulders. In this manner the proper position of the head is maintained until fusion becomes firm.

Constant nursing of the patient is imperative until he is fully conscious, lest the head be moved during the interval when he is recovering from the anesthetic. Even during the first few days postoperatively the patient can be turned from one side to the other at frequent intervals, traction on the head and the sand-bags, of course, being removed before turning. In this manner nursing care of the patient is facilitated, and the patient is made more comfortable. After two weeks the patient can be placed on his side for longer periods.

OPERATIVE FINDINGS

Among the more frequent findings which were noted at operation was the formation of scar tissue, and in a few instances old extravasated blood was found in the soft tissues about the spine posteriorly. Impingement of spinous processes and laminae was noted in 5 cases. In 7 cases ununited fractures of the posterior elements were found. In most of these the fractured portion of the vertebral arch was freely movable. Hypermobile luxated vertebrae were encountered in 6 cases. Complete bilateral dislocation of the articular facets was noted in 4

cases, while a severely crushed articular process was found in another case. Natural fusion of impinging laminae was present in 1 case. Frequently the laminae of the vertebra involved were displaced anteriorly, and in 3 instances these laminae were removed because they encroached on the cord. In 1 instance moderate anterior displacement of a lamina had caused an indentation on the cord.

POSTOPERATIVE COURSE

Little evidence of shock or other complications were present in these cases, and the immediate postoperative reactions were, as a rule, of a mild degree. An exception to this statement was a patient in whom

Fig. 4.—Postoperative roentgenogram showing solid bony fusion of the posterior elements while the involved vertebral body remains without bony ankylosis.

severe hemiplegia developed after spinal fusion, and further surgical treatment was later necessary. Slight edema of the occipital region and of the face was encountered postoperatively in 2 cases, and a generalized weakness of the extremities was present in 2. These complications lasted for only a few days, and no permanent damage was done to the cord. In a few cases there was a slight degree of abdominal distention, and in 2 cases urinary retention was present. These complications gave no alarm and were transient.

During their stay in the hospital all these patients, with the exception of those on whom further surgical measures were necessary, showed some degree of improvement in comparison with their preoperative status. The greatest and most constant improvement was in the amelio-

ration of symptoms arising from the vertebral pathologic process. Symptoms arising from involvement of the nerve roots were slightly improved but with little change in comparison to the preoperative condition. One patient with severe involvement of the spinal cord was greatly improved, while 2 with severe involvement were only slightly improved. Control of the sphincter muscle, however, had returned, and the patients were able to get about with the aid of crutches, whereas before they entered the hospital they had been bedridden.

Fig. 5.—Postoperative roentgenogram showing solid bony fusion of the posterior elements and bony ankylosis of the involved vertebrae.

With the exception of the 1 patient who came to secondary operation two weeks following the primary operation, the remaining 17 were allowed out of bed, with no supportive apparatus, in from six to twelve weeks postoperatively. These patients were discharged from the hospital wards in from seven to thirteen weeks postoperatively. The remaining patient in the series was discharged one year postoperatively. During the stay in the hospital this patient was in bed continuously.

FOLLOW-UP STUDY

The longest postoperative follow-up period in this group was six years and the shortest two months, while the average period was two years. After discharge from the wards the patients were seen at fairly frequent intervals in the dispensary. Almost immediately after discharge physical therapy in the form of massage of the cervical region, along with active and passive motion of the cervical portion of the spine, was started. Muscle training and reeducation of partially paralyzed groups of muscles were also carried out for a long period, while various mild contractures were dealt with by the aforementioned types of therapy as well as stretchings.

After spinal fusion of the cervical region in which two or more vertebrae are fused, one would naturally suppose that the mobility of the head would be limited to a considerable degree. However, this was not the case in this series, for 13 of the 18 patients presented free, unrestricted and painless motion of the head. In 5 instances there was a mild to moderate degree of limitation, mechanical rather than due to pain or spasm. One patient complained that he suffered from fatigue of the posterior cervical region. This would develop usually after he had been sitting for from three to four hours. In this case there was only 20 degrees extension of the head, and the patient held his head in a slightly flexed attitude. In this instance a portion of the cervical segment of the spine had been fused in a slight degree of flexion, causing an unnatural attitude of the head.

Fusion was reported as solid roentgenographically in 12 cases in from six to twelve weeks after operation. Postoperative roentgenograms were not made in 3 cases, while in 2 instances pseudarthrosis developed. Another patient required further surgical treatment a few weeks after the first operation had been performed. In those cases in which pseudarthrosis developed, repair was not attempted because neither of the patients had sufficient trouble to justify it.

By relieving these patients of preoperative vertebral symptoms of pain, crepitus and spasm, spinal fusion enabled them to move the head more freely than before operation. Also, the feeling of stability which is experienced after fusion of the spine dispels the fear which those patients had of moving the head.

Fourteen patients presented sufficient evidence preoperatively to be classified as having involvement of the nerve roots. Seven had moderate involvement and 7 mild. Two of those who had moderate involvement and 4 of those who had mild involvement preoperatively showed no evidence of irritation of the nerve roots at the follow-up examination, while 5 of those who had moderate involvement preoperatively showed involvement of a mild degree at the follow-up examination, and 3 of those who had mild involvement showed no change.

Ten cases in which preoperative and postoperative roentgenograms were available have been reviewed in connection with the relationship between anterior vertebral displacement and tilt in the presence of involvement of the nerve roots. Five of the patients had anterior displacement of the involved vertebra of from 20 to 8 per cent. Four of these had moderate irritation of the nerve roots preoperatively, while 1 presented no evidence of involvement. On their last visit to the dispensary 2 of the patients were without a nerve root syndrome, while 3 had a mild syndrome. Of 5 others with anterior vertebral displacement of from 0 to 20 per cent before and after operation, 1 had moderate and 4 had mild involvement of the nerve roots preoperatively, while postoperatively and on their last visit to the clinic, the patient with moderate involvement and 3 of those with mild involvement were relieved, while 1 patient still had a mild degree of irritation of the nerve roots.

Seven patients had anterior vertebral tilt of the involved vertebra of from 20 to 58 degrees both preoperatively and postoperatively. Four of these had moderate involvement of the nerve roots preoperatively, while 2 had mild involvement, and 1 had no evidence of injury to the nerve roots. After fusion, the moderate involvement in 2 patients decreased to a mild degree, and 2 of the patients presented no evidence of involvement. The 2 patients with mild involvement preoperatively had no evidence of injury to the nerve roots postoperatively, and the remaining patient was without any involvement, as before operation.

Three other patients had an anterior tilt of the involved vertebrae of from 0 to 20 degrees. One presented evidence of moderate involvement and 2 evidence of mild injury to the nerve roots. The patient who had moderate involvement preoperatively had mild involvement postoperatively; the condition of the patient with mild involvement remained the same, while the other patient did not show any evidence of involvement.

After operation and while these patients were in the hospital there was a definite improvement in the degree of irritation of the nerve roots in some cases. The improvement in the nerve root syndromes was gradual and steady, but after a few months little change was noted.

In comparing preoperative and postoperative roentgenograms, the percentage of anterior displacement and the degree of anterior vertebral tilt in these 10 cases did not change to a great extent. In some instances there was a decrease in the degree of tilt or displacement, while in others an actual increase had taken place. From the fact that so many of these patients were relieved of their nerve root syndrome by spinal fusion, and yet little or no correction of these deformities had taken place, one must naturally suppose that vertebral tilt of a moderate

degree, as well as vertebral displacement, if not accompanied by mobility, plays little part in the production of a nerve root syndrome in this type of lesion in its chronic stage.

Six of the patients in the series had a hypermobile luxated vertebra. Two of the patients had moderate involvement of the nerve roots preoperatively, while postoperatively 1 was without involvement, and the other had a mild degree. Two other patients with hypermobile luxated vertebrae had a mild degree of involvement preoperatively and none postoperatively. The remaining 2 patients had neither preoperative nor postoperative involvement. The number of hypermobile vertebrae in this series is small, and yet it may be misleading since the data in regard to gross pathologic changes at operation were very sketchy and incomplete. However, it is interesting to find that 4 of 6 patients with involvement of the nerve roots and with a hypermobile luxated vertebra were either completely or partially relieved of that involvement by spinal fusion. Perhaps the involvement of the nerve roots, which one so often finds as a feature in lesions of the cervical segment of the spine in the chronic stage, may well be explained by the presence of a hypermobile luxated vertebra. Certainly a very mobile vertebra, which is also luxated, can cause a strain on its nerve roots, while the elimination of motion of that vertebra by spinal fusion will produce a mitigation of a syndrome arising from involvement of the nerve roots. It might be assumed that patients in this series who presented such a syndrome but were not aided by spinal fusion did not have a hypermobile vertebra and that sufficient damage to the nerve roots or their coverings had been done at the time of the primary trauma to leave them in a chronically impaired state.

INVOLVEMENT OF THE CORD

Spinal fusion alone was done on 4 patients having involvement of the cord. One of these patients had mild preoperative and postoperative involvement, while another had mild preoperative involvement and severe postoperative involvement. The latter was the one with the severest displacement and tilt of the entire series, and there is no doubt that if, in the presence of such a degree of deformity, laminectomy had been done in conjunction with spinal fusion a far more desirable result would have been obtained. Moderate involvement was present in 1 patient preoperatively and mild involvement postoperatively, while another patient had mild involvement before operation and none after operation.

A one stage laminectomy and spinal fusion were carried out on 5 patients. Two of these had neither preoperative nor postoperative evidence of a syndrome, the laminectomy being done on 1 to remove

a very mobile fractured vertebral arch which at any time could have caused severe damage to the cord, while laminectomy was done on the other patient to remove a severely displaced vertebral arch. Laminectomy and spinal fusion were done on 3 other patients with severe involvement of the cord. In 1 of these there was a decided improvement in the cord syndrome, while 1 was relieved to a moderate degree, the remaining patient being slightly improved but still having severe involvement.

As has been mentioned before, the dura was incised at the primary operation in only 1 instance, and yet it was observed in many cases that the dura was thickened. I believe that in the presence of involvement of the cord, when laminectomy is done, the dura should be opened freely if it is at all thickened. An unyielding dura can cause as much

TABLE 3.—*Preoperative Status of Patients*

Case	Vertebral Syndrome	Nerve Root Syndrome	Cord Syndrome
1	None	Mild	Severe
2	None	None	None
3	None	Mild	None
4	None	None	Mild
5	None	None	None
6	None	None	None
7	None	Mild	Mild
8	None	None	None
9	None	None	None
10	None	Mild	None
11	None	Mild	Mild
12	None	None	None
13	None	None	None
14	None	None	None
15	None	None	None
16	None	Mild	None
17	None	Mild	Moderate
18	None	Mild	Severe

pressure on the cord as a pressing vertebral arch, especially in the presence of anterior vertebral displacement or a kyphos.

Laminectomy in 1 case caused a decided improvement in the cord syndrome. By this procedure a severely displaced and fractured vertebral arch was removed. This had caused pressure on the coverings of the cord and on the cord. Neither the presence of a pressing arch nor a fracture of the arch was diagnosed preoperatively.

DISABILITY

The degree of disability of the patients in this series is based on the severity of the cord, nerve root and vertebral syndromes. Preoperatively 3 patients had a severe degree of disability, 13 a moderate degree and 2 a mild degree. Postoperatively, 2 of the patients had a severe degree of disability, 1 a moderate degree and 6 a mild degree, while 9 were without any form of disability. In other words, the results of treatment in 10 cases may be classified as excellent. Nine of the

10 patients having no cord, vertebral or nerve root syndromes had a normal degree of motion in the cervical portion of the spine. One of the patients presented severe involvement of the cord preoperatively, while postoperatively the involvement was of a mild degree. The results in 5 cases were classified as good, i. e., a marked decrease in the severity of the syndromes had taken place. The results in 1 case were fair, while in 2 they were poor. In 1 of the latter cases the patient was much worse than before operation, while in the other case the patient was improved slightly but was still severely handicapped.

In table 3 is given the postoperative status of each patient.

CONCLUSIONS

1. Closed reduction of a fracture-dislocation or dislocation of the cervical portion of the spine in many instances fails to restore the patient to normal.

2. The disability of the patient should be estimated in regard to the degree of involvement of the vertebral column, the nerve roots or the cord.

3. The type of involvement of the cord is difficult to ascertain in a case of a lesion of this nature.

4. An ununited fracture of the vertebral arch is common in such cases and is difficult to diagnose.

5. Natural fusion of the vertebral bodies of the involved vertebra does not occur in a number of cases.

6. Hypermobility of the luxated vertebra is the cause of involvement of the nerve roots in many cases.

7. Spinal fusion of the cervical region is not a dangerous procedure in experienced hands.

8. Spinal fusion is of little value in the treatment of syndromes arising from involvement of the cord, but if possible a combined laminectomy and spinal fusion should be attempted in the presence of involvement of the cord and anterior vertebral displacement.

9. If laminectomy is done, the coverings of the cord should be freely opened if they are at all thickened.

10. Spinal fusion relieves a patient of symptoms referable to the vertebral pathologic processes and is the only adequate means to stabilize a hypermobile luxated vertebra.

11. The mobility of the head and neck is not materially limited by fusion of three or four cervical vertebrae.

12. Anterior vertebral tilt and displacement without mobility play little part in the production of nerve root syndromes.

CLEFT PALATE

A CORRELATION OF ANATOMIC AND FUNCTIONAL RESULTS FOLLOWING OPERATION

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The operation for cleft palate was presented for the first time comprehensively, according to Dorrance, in 1816 by von Graefe, the founder of modern plastic surgery. The operation which is known today as the Langenbeck procedure was described in 1861 by von Langenbeck.

An extensive literature has accumulated since these early operations for cleft palate, and but a small amount of it pertains to the functional results after treatment.

Reports on the phonetic outcome in a group of cases may be satisfactory individually, but great difficulty is encountered in comparing the results in one group with those in another. The difficulties of judging the phonetic results of other investigators arise because, in the first place, the observations made for each report depend on the auditory sense, which is individually variable. Second, there are so many factors involved in speech that a method of anaylsis satisfactory to one observer might not meet the demands of others. Third, the problem of a foreign tongue makes the task of comparison more difficult, for it is probable that certain languages may be more perfectly spoken by a person with a cleft palate.

Ideally, the most accurate evaluation of phonetic results can be obtained only by a trained phoneticist. However, this ideal is difficult to obtain.

The main purpose of this work is to correlate the speech defect with the anatomic result and to make the observations in as simple a manner as possible and by a method which can be carried out by one with no special training in phonetics.

REVIEW OF THE LITERATURE

Whitehead first tabulated the phonetic results in such cases in 1870. Of five patients who had undergone repair for a cleft palate, one had

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distinct speech, two showed improvement in speech, one had "intelligent speech" but with some nasalization and one wore an obturator.

Bond stated in 1893 that he had never seen a person with a corrected cleft who did not have some speech difficulty.

Kappeler in 1902 stated that it is rare to obtain normal speech without training. Of sixteen patients who did not receive training and did not use an obturator, four had normal speech, one showed noticeable improvement and eleven showed no improvement. Of thirteen who received speech training only, eleven had normal speech, and two showed noticeable improvement. Of twenty-nine who received speech training and also used an obturator, twenty-three had normal speech, and six showed noticeable improvement.

Church in 1904 disagreed with the widely accepted opinion that distinct speech could be acquired by a person with an unrepaired cleft palate.

In 1904 Ranzi and Sultan reported the results in one hundred and one cases. In six, speech was normal; in forty-four, there was improvement; in fourteen, the functional result was not successful; in twelve the operation was unfinished, and in twenty-five, the results were not known.

Berry reported the results in two series of cases. Of the first series of sixty-one cases, reviewed in 1905, he stated: "In no case has speech after operation become absolutely normal, and there are comparatively few in which I should call it very good." According to his data, speech was very good in five cases, good in eighteen, fair in fourteen, indifferent but improving in three, bad in four, already much improved in eight and not yet improved in four. Three of the patients had not yet learned to speak, and no report had been received concerning eight.

In the second group of eighty-one cases, reviewed in 1911, he stated that there were some cases in which speech was so nearly perfect that it was difficult to detect anything wrong. The results are tabulated separately for individual cases. An analysis shows that the results are apparently more favorable than those in the previous series.

Brown in 1908 stated that the ultimate result is measurable only by the perseverance by which continued speech training is received. He noticed that the parents of many children with cleft palate had some speech defect which may have been either a nervous inefficiency in speech mechanism or a slight imperfection of anatomic development, either of which became a cleft in the next generation. He also stated that scars of the lips disturbed the articulation of b, f, m, p, v and w and that in many cases the articulation of c, d, g, h, j, k, l, n, r, s, t and z was poor because all of these sounds, which are uttered by the tongue behind the teeth, were distorted by an imperfect arch and teeth caused by ill advised operations in infancy.

Blakeway stated, in comparing results of different operations in 1914, that of seventeen persons operated on by the Lane teechnic there were five who were old enough to speak, and all of these were poor speakers.

Brophy stated in 1914 that "if operation is properly performed early, no defect is noted in phonation." He also stated that if operation is performed between the ages of 2 and 12 more or less defect in speech will remain unless the patient is properly trained by one who understands speech.

Stahl in 1923 reported the following results in thirty-one cases: In ten speech was perfect; in eight, good; in seven, fair; in two poor, and in four, very poor.

Monnier reported the results in ninety-three cases in 1924. In twenty-two speech was good; in thirty-four, good with slight nasalization; in fifteen, distinct with marked nasalization, and in twelve, indistinct, while in ten the patient had not yet learned to speak.

Barrington-Ward in 1927 reported that speech was good in twenty of fifty-seven cases, fair in eighteen and bad in twenty. Four of the persons with bad speech were mentally defective.

Tschmarke reported normal speech in 9.1 per cent of forty-five cases, improved speech in 57.6 per cent and unimproved speech in 33.3 per cent. He presented the following tabulation in which are compared the percentage of well healed palates and the satisfactory functional results obtained by different authors:

	Healed Palates	Satisfactory Functional Results
Author	%	%
Kappeler	86.5	53.3
Ranzi and Sultan	69.3	9.3
Stahl	58.7	15.0
Veau and Ruppe	74.0	25.0
Dreher	60.9	34.7
Tschmarke	64.3	9.1

Turner stated that of forty-four cases in which the palate was completely restored, speech was normal in seven, good in twenty, fair in fourteen and bad in three.

Halle stated that in most instances if operation is performed in the first year speech will be normal or almost normal.

Nitch in 1927 obtained good speech in thirteen of thirty-five cases, poor speech in seventeen and bad speech in five.

Burdick in 1930 remarked that no matter how successful an operation is and no matter what type of operation is performed the speech will be far from normal. He obtained good results in nine of twenty-three cases of incomplete cleft and in six of twenty-two cases of complete cleft.

Pagnamenta in 1932 tabulated his anatomic and functional results. These are presented in table 1.

In 1929 Veau reported the phonetic results in one hundred cases cases in which the classic procedures of Langenbeck or one of its modifications was used. The speech of ten of the patients improved rapidly, and they spoke well or nearly normally; the speech of six was improved by phonetic education and presented little, if any, defect. Twenty-four of the patients had poor speech with little intelligibility.

In 1922 Veau reported the ultimate functional results of forty in which his procedure was used. Thirty-five per cent of the patients had normal phonation; 29 per cent had good speech but with nasal air and bad nasalization, and 36 per cent had vocal deficiency in that they were unable to produce certain sounds such as p, t and k or ch, s, j and z.

In 1933, in a second series of one hundred cases, he found 62 per cent of the patients with normal speech, 23 per cent with the nasal air and nasalization and 15 per cent with vocal deficiency.

TABLE 1.—*Anatomic and Functional Results of Pagnamenta*

Type of Cleft	Number of Cases	Good Palates, %	Understandable Speech, %	No Nasalization, %	Slight Nasalization, %	Severe Nasalization, %
Partial cleft	27	55	77	29	14	5
Subtotal cleft	48	39	63	21	46	33
Total unilateral cleft with lip defect	54	30	55	15	43	42
Total bilateral cleft with lip defect	16	23	42	0	35	65

Throughout all his work Veau stressed the fact that a good anatomic result is essential for a good functional result regardless of speech training. Of one group of forty patients with normal speech, twenty-four had had no speech training. Of the sixteen who had to be reeducated, fifteen spoke before operation.

Other articles, by Veau and Borel, and Ruppe and Ruppe, give their methods for analyzing and training of speech.

MECHANISM OF CLEFT PALATE SPEECH

There are two main characteristics of speech which are noted in practically all cases of cleft palate: (1) disturbed quality of tone and (2) disturbances of articulation.

1. A disturbed quality of tone is exemplified by what is commonly known as "nasalization," "nasaling," "nasal tone" or "talking through the nose." The term nasalization will be used in this analysis. It is also exemplified by a lack of what is usually termed "resonance" and also of "inflection," thereby giving the flat, uninteresting monotone so characteristic of the person with a cleft palate.

The following is an interpretation of this disturbed quality of tone after a study of the current texts on speech and voice:

After a column of air which has been put into motion by the lungs, intercostal muscles, diaphragm and abdominal musculature passes through the larynx, it emerges with a sound which has two characteristics, pitch and intensity.

The pitch is determined by the number of vibrations of the vocal cords and is the result of a fundamental tone plus a range of overtones or undertones, called partials. Ordinarily the ear is not aware of these partials, as they, together with the fundamental tone, are heard as one sound with a definite pitch.

When the column of air, which now has pitch and intensity, reaches the pharyngeal, oral and nasal cavities, it enters into "resonating" cavities. These cavities have the power to set up vibrations which differ according to their size and shape. The number of vibrations may equal the vibrations of the fundamental tone, in which case this tone is augmented; or the number of vibrations may equal those of one of the partials, in which case that particular partial is augmented, or a number of different vibrations may be set up at the same time augmenting a combination of partials. The result of this modification of the original tone by the vibrations set up in the upper respiratory passage adds a third characteristic to the original tone, a quality or timbre.

This addition of quality is well demonstrated by Bell:

> Press your lips firmly together while you blow air between them, so as to cause the edges to vibrate. The sound produced is not very pleasant and resembles, more than anything else, the hum of a bee . . . . But place the buzzing lips at the end of a tube, for example, a trumpet, and at once the quality changes . . . . In this case the source of the sound is found in the vibration of the lips but the timbre or quality is due to the trumpet.

Any change in the resonating cavities of the upper respiratory passages will change the quality of a sound.

There are three main defects in these cavities in cases of cleft palate which tend to change the quality of a sound.

(*a*) The first is defective closure of the velopharyngeal sphincter. This defect permits the column of air to be shunted away from the normal resonating chamber, the mouth, to an abnormal one, the nose. It not only gives the flat monotone speech characteristic of a person with a cleft palate but also is the cause of nasalization. This deformity is by far the most serious of the three defects and the resulting quality of sound is called rhinolalia aperta.

(*b*) Changes in the size and shape of the chambers, giving an abnormal nasal resonance, is the second cause of defective quality of speech. Nasal resonance of some degree is a part of all normal speech and is acquired by vibrations set up in the nasal cavities by the column of air below the closed velopharyngeal sphincter trans-

mitting its impulses through the soft palate, thus adding a nasal quality to the resultant tone. That the patient with a cleft palate usually has a nasal passage of abnormal size and shape is commonly recognized. This abnormality is most frequently a narrowing of the nasal cavities, by a deformed septum, enlarged turbinates or enlarged adenoids. The resulting defect in quality of sound is called rhinolalia clausa and is exemplified in a person who has a cold.

Cleft palate speech, which is usually characterized by both of the aforementioned defects in quality, rhinolalia clausa and rhinolalia aperta, is designated as rhinolalia mixta.

(*c*) The third anatomic defect which may disturb the quality of a tone is less important but must play some rôle, although it is impossible to analyze it separately. This defect consists of the scar tissue and taut mucous membrane surfaces which result from the operation and which undoubtedly hinder the vibrating property of these surfaces.

2. Disturbances of articulation, the second characteristic of speech noted in the majority of cases of cleft palate, has been well summarized by Hudson Makuen. According to him, normally consonants are made

> . . . by impeding the column of air of breath at certain points above the larynx. The points at which this impediment takes place are called the stop positions. These have been divided into the anterior, middle and the posterior stop positions. The anterior one is formed by the lips (in the articulation of the so-called labial sounds, p, b, m, wh, w), by the lower lip and teeth (in the articulation of the labiodentals f and v) and by the tip of the tongue and teeth (in the articulation of the linguo-dentals th and ch); the middle one by the tongue and the hard palate (in the articulation of the anterior-linguo palatals s, z, sh, t, d, n, l); and the posterior one, by the dorsum of the tongue and soft palate (in the articulation of the posterior linguopalatals r, g, h, y). For all these sounds requiring an impediment in the outgoing column of breath, whichever stop position may be used, it is necessary to have a freely movable and normal palate.

In the cleft palate which has not been repaired, the anterior and middle stop positions are rarely used because of the inability of the patient to focus his vocalized breath at this point when there is a free passage for air through the nasal cavity. The patient uses substitute stop positions, such as the anterior nares, the base of the tongue and the posterior pharyngeal wall and the lips of the larynx formed by the arytenoid cartilages and the arytenoid and aryepiglottic folds.

When perfect closure of the velopharyngeal sphincter is obtained after operation, this habit of using substitute stop positions is one of the major causes of poor speech.

ANATOMIC CONSIDERATION

Dorrance, Whillis and Wardill have presented this subject with special emphasis on the sphincteric action of the muscles of the soft palate and the posterior pharyngeal wall.

The "velopharyngeal sphincter" is a well established entity, and hereafter insufficient approximation of the soft palate with the posterior pharyngeal wall will be described as velopharyngeal insufficiency.

MATERIAL AND METHOD OF USE

This report concerns one hundred cases of cleft palate in which an operation was done for repair.

The records of seventy-five cases were obtained from the files of Dr. H. P. Ritchie. Those of thirteen cases were obtained from the files of the Special Class Department of the Minneapolis Board of Education and concern children who had had speech training in the city schools at one time or another. The data on the remaining twelve cases were obtained at the University of Minnesota Hospital Dispensary as the patient returned for a routine check on his condition. All patients observed were used for this study except a few who were too young to cooperate or those who refused to talk.

The greater portion of the patients were operated on by Dr. H. P. Ritchie or by one of his associates. The type of operation has not been recorded in the study of each case. However, the majority of persons were operated on by the fundamental Langenbeck procedure with suturing of raised mucoperiosteal flaps in the midline, muscle suture of the soft palate with a figure of eight suture and occasional wiring of the premaxilla, as described by Ritchie. In the last few years the wiring of the alveolar process has been discontinued, except in cases in which the defect is of unusual degree, and instead the lip is repaired as a preliminary procedure to mold the alveolar process by muscle pull.

The sounds and words used in the phonetic outline have been suggested by Dr. Bryngelson, of the University of Minnesota Speech Clinic, and are taken from his outline on articulatory speech disturbance, which is a modification of the Travis-Rasmus speech sound discrimination tests.

All of the cases were grouped in the manner suggested by Davis and Ritchie in 1922, which is briefly:

Group I. Lip cleft.

Group II. Palate cleft (normal lip and alveolar process), designated according to the length of the cleft of the hard palate as ⅓, ⅔ or ⅔ and cleft of the soft palate only.

Group III. Lip, alveolar process and palate cleft, designated as right, left or bilateral and complete or incomplete, i. e.:
Right complete or right incomplete
Left complete or left incomplete
Bilateral complete or bilateral incomplete

TABLE 2.—*Outline Used For Analysis of Phonetic Results After Operation in Cases of Cleft Palate*

Father's name..... Address.....
Patient's name..... Birth date.....Age.....
Patient's group..... Hospital and record number.....
Phonetic education.....

I. Lip:
Form..... Operation date..... Interval.....
Examination: 1. Nostrils: Contour.....
2. Body of lip: Looks..... Motion.....
3. Speech:

Letter	Initial	Medial	Final	Result
O	old			.....
	so			.....
	The old oak stood so much alone			.....
U	unit			.....
	use			.....
	Few argue and use cues as fuel			.....
B	base	may be	cab	.....
	Baby Bee bit her boots			.....
P	pay	paper	ape	.....
	Paul slipped on a potato peel			.....
M	May	camel	same	.....
	May made mama maim the man			.....
V	vain	ivy	save	.....
	Veal was divided in five parts			.....

Comment: .....

II. Process:
Form..... Operation date..... Interval.....
Wires or lip or both.....
Examination: 1. Defect present.....
2. Speech:

Letter	Initial	Medial	Final	Result
T	tail	city	sit	.....
	Tom ate ten tomatoes			.....
D	do	daddy	fade	.....
	Fido is Freda's poodle			.....
Ch	chart	catcher	patch	.....
	The pitcher changed his church			.....
Th	then	others	path	.....
	Then the path of others was strengthened			.....

Comment: .....

III. Palate—Hard:
Form..... Operation date..... Interval.....
Examination: 1. High..... Median..... Low.....
1. Speech:

Letter	Initial	Medial	Final	Result
Y	year	yoyo		.....
	Yale men yelped at the Yeoman			.....

Comment: .....

IV. Palate—Soft:
Form..... Operation date..... Interval.....
Examination: 1. Anatomy:
a. Uvula.....
b. Motility: 1..... 2..... 3..... 4.....
c. Texture: Full..... Soft.....
Thin..... Hard.....
d. Approximate length..... Long.....
Median.....
Near.....
e. Approx. dist. to post. pharyngeal wall..... Long.....
Median.....
Near.....
f. State when "Ah"
Approx. dist. to post. pharyngeal wall..... Long.....
Median.....
Near.....
Superior constrictor muscle.....
Well developed.....
Undeveloped.....
Insertion of levator.....
Prominent.....
Not prominent.....

TABLE 2.—*Outline Used For Analysis of Phonetic Results After Operation in Cases of Cleft Palate—Continued*

2. Functional:
   a. Functional test.....
   .....
   b. Speech:

Letter	Initial	Medial	Final	Result
K	cape	ache	racket	.....
	Keep cool if you can			.....
G	get	agony	lag	.....
	The rogue stole the goose			.....
Ng		slinging	hang	.....

Comment: .....
.....

General Impression: Anatomic Result:
Lip.....
Process.....
Palate (hard).....
Palate (soft).....
Functional Result.....
.....

Table 2 presents the type of outline used in each case.

It has been the purpose in this study to follow the suggestion of the classification of Davis and Ritchie and take up each cleft separately, viz., the lip, the alveolar process, the hard palate and the soft palate, and to choose sounds which are made for the most part by these structures and from which any deviation from the normal can easily be ascertained.

For the lip itself, b, p, and m are used. O, u and v were also used, although not true labials, because the lip is used in a different manner than b, p and m.

For the alveolar process, sounds classified not only as linguodentals, such as ch and th, but as anterior linguopalatals, such as d, t and s, were used. The latter are used in the test for the alveolar process instead of for the hard palate because they are articulated at the junction of the alveolar process and the hard palate.

For the hard palate, y (j) was the sound used. Reference was also made to the anterior linguopalatals of the preceding group.

For the soft palate, k and hard g as well as ng were used. In this analysis the anatomic study was more comprehensive. The motility was graded in degrees from 0 to 4, the latter being that of a normal palate.

No accurate measurements by linear scales were made. Estimations of the length of the repaired palates were recorded. After fifty of these had been recorded, the average length was estimated. Those palates of average length were recorded as normal; those longer than average were recorded as long, and those shorter than average were recorded as short.

The same procedure was carried out for the estimations of the distance from the palate to the posterior pharyngeal wall in the quiescent state and also when "Ah" was said.

Comparisons were not with the normal palate. Similar estimations on twenty-five normal palates indicated that the average length of the normal palate was longer than that of the cleft palate, and the average distance to the posterior pharyngeal wall both in the active and in the quiescent state was shorter in the normal than in a cleft palate. Consequently, when a cleft palate was designated as long, it was long as compared to the average length of a group of cleft palates and not to the length of a normal palate.

The consistency of the soft palate as well as the development of the superior constrictor muscle and the prominence of the insertion of the levator muscles were also recorded.

The functional test used in the majority of cases was performed as follows: A small piece of rolled paper was placed on the surface of each of two pieces of cardboard, measuring about 3 by 2 inches. One of these was held just above the vermilion border of the upper lip and the other just below the vermilion border of the lower lip. The patient was then told to blow the piece of paper off the lower one. If any air escaped through the nose, the piece of paper on the upper cardboard also moved or was blown off the card, showing that there was incompetent closure of the velopharyngeal valve.

ANALYSIS OF MATERIAL

The purpose of this analysis, as previously stated, is to determine to what degree and manner the anatomic defect influences the functional result after operation.

One hundred cases were studied. Seventy-seven were classified as belonging to group III. In fifteen of these the cleft was bilateral, and in sixty-two, either right or left unilateral. In two of the fifteen cases of bilateral cleft the palate was normal. Twenty-three cases were classified belonging to group II. Twenty of these were placed either in group II 1/3, group II 2/3 or group II 3/3. Only three were classified as belonging to group II, cleft of the soft palate only.

The sounds used for the analysis were the consonants, except in one instance, in which o and u were used. The phonetic defect of these consonants is usually made up of two elements. The first is a result of insufficient closure of the velopharyngeal sphincter, resulting in nasalization. The second is the articulatory defect, which may be the result of nasalization but to which is added the defect caused by an anatomic fault at a stop position.

In every case an attempt was made to establish the relative importance of insufficient closure of the velopharyngeal sphincter and the anatomic stop position.

Correlation as Regards Defects of the Lips.—The lip is the labial portion of the anterior stop position. B, p, m, v, o and u were the sounds used. This part of the study included only the cases in group III, in which the clefts were bilateral or unilateral and complete or incomplete. In none of the cases in group II was there an anatomic defect of the lips.

Whether the phonetic defect was in the initial, medial or final stop position is designated by numerals 1, 2 and 3. A defect of an initial p is designated as p 1, of a medial p as p 2 and of a final p as p 3. The same procedure is used throughout for all the stop positions, whether the lips, the alveolar process or the palate are being studied.

An analysis of the phonetic results in those cases in which there was a defect of the lip is given in table 3. A discussion of this table follows:

B 1 was absent in one case (67) and faint in another (21), while p 1, 2 and 3 was omitted in one case (37). There was some degree of nasalization in all these cases, and as the main cause for omission of sounds is inability to focus the air on the correct stop position, due to imperfect closure of the velopharyngeal sphincter, this nasalization rather than the anatomic defect of the lip can be held responsible for the phonetic defect.

B 1 and p 1 were articulated through the nose in one case (33). There was no anatomic defect of the lip in this case, but nasalization of 2 degrees was the cause of the phonetic defect.

U 1 was slightly indistinct in one case (15). The function of the lips was good in this case, but there was nasalization of 4 degrees, which in all probability caused this defect.

The most common defect was the substitution of b for p, m for p or m for b. In all of these cases except case 29, there was some degree of nasalization. With nasalization present, the tendency is to use the sound which is made by a similar architecture of the stop position but which requires less perfect closure of the velopharyngeal sphincter.

In one case (29) there was no anatomic defect of the lip and no nasalization, yet the patient substituted m for b 1. The record of this case showed that the patient talked perfectly if the speech was slow and that the phonetic defect occurred only when the speech was rapid. This demonstrates the occasional case in which there is perfect anatomic function but the patient has not developed the power to move the soft palate rapidly.

In view of the results of this analysis, the conclusion is that as far as the labial element of consonants is concerned the patients in group III can adequately compensate for any anatomic defect of the repaired lip.

Correlation as Regards Defects of the Teeth, Alveolar Process and Extreme Anterior Part of the Hard Palate.—The linguodentals, such as th, ch and s, and the anteriolinguopalatals, t and d, were employed in this analysis. There were four types of functional defects and fourteen variations of anatomic defects. The number of functional defects outnumbered the anatomic defects, as there were two separate types of functional defects in twelve cases. Thus the totals have no reference to the number of cases (table 4).

1. Slur of ch or s or both. The recording of this defect was the most difficult of all, for if one critically analyzes a group of normal

TABLE 3.—*Analysis of Functional Defects in Relation to Anatomic Defects of the Lips*

Case	Anatomic Defect	Functional Defect	Nasalization, Degree
9	Short lip	None	0
13	Short lip	None	0
23	Short lip	None	0
34	Short lip	p 1 = b 1	3
57	Short lip	None	2
15	Abnormal scar	u 1, slightly indistinct	4
22	Abnormal scar	None	2
24	Abnormal scar	b 1 = m 1	1
40	Abnormal scar	None	2
55	Abnormal scar	None	—1
63	Abnormal scar	None	1
20	Muscle out of contact	None	2
62	Muscle out of contact, large hole in hard palate also	b 1 = m	1
26	Tight lip	None	1
50	Tight lip	None	1
37	Notch in vermilion border	p 1, 2, 3 omitted	1
46	Notch in vermilion border	None	0
53	Notch in vermilion border	None	0
41	Poor motion	None	0
67	Poor motion	b 1 = m, b 1 omitted	3
8	No anatomic defect	p 1 = b 1	1
21	No anatomic defect	b 1, faint	1
29	No anatomic defect	b 1 = m	0
33	No anatomic defect	v 1 = b; b 1 and p 1, through the nose	2
60	No anatomic defect	b 1 = m	3
61	No anatomic defect	b 1 = m	2
93	No anatomic defect	b 1 = m	1

persons, the probabilities are that a slight slurring element will be found in many of them on the articulation of the sibilants.

In forty-two of the sixty-five cases, or 64 per cent, there was some degree of slurring of ch or s. In some cases it was extremely difficult to determine whether there was a sufficient degree of slurring to class it as a defect, but if there was the slightest question it was recorded as such.

This defect, although the most frequent, is by far the least important as far as the general speech of the patient is concerned. In fourteen of the forty-two cases, or 25 per cent, in which the results in regard to speech were ultimately classed as excellent, this defect of slurring of ch or s was so slight that there was no hesitation in classifying the results as excellent.

The question as to whether the anatomic defect of the stop position has more influence on this functional defect than the degree of velopharyngeal insufficiency is difficult to determine.

The fact that in thirty of the forty-two cases, or 71 per cent, some degree of velopharyngeal insufficiency was present would lead one to conclude that the latter element is an extremely important one.

On the other hand, it was noted that the degree of nasalization was minimal in practically all of these cases. As only a moderate force is needed to articulate a good ch or s, it is quite possible that perfect closure is not absolutely necessary for a good ch or s and that a minimal degree of nasalization plays no rôle in slurring of ch or s.

TABLE 4.—*Incidence of Functional and Anatomic Defects*

Functional Defects	Incidence
1. Slur of ch, s, or both	42
2. Ch or s or both spoken through the nose	10
3. Omission of s, ch 1, 2, 3; d 1, 2, 3 or t 1, 2, 3	15
4. Substitution: d = n, t= d = n, s = n	5
5. Absence of functional defect; anatomic defect	5
Total	77
Anatomic Defects	
1. Atrophied premaxilla	2
2. Malocclusion	4
3. Notch in alveolar process	4
4. Orthodontia	8
5. Teeth out of line	16
6. Tooth misplaced posteriorly	5
7. Tooth misplaced anteriorly	1
8. Teeth abnormally spaced	4
9. Small hole anterior to alveolar process	8
10. Overlapping of medial with lateral process	4
11. Overlapping of medial by both lateral processes	1
12. Teeth in poor condition	1
13. Short process, teeth out of line	2
14. No teeth present	1
15. Absence of anatomic defect; functional defect	4
Total	65

Furthermore, the contour of the teeth plays an important rôle in the articulation of the sibilants, and as the greatest number of cases of defective articulation were observed in persons in whom the contour of the teeth was defective, the probabilities are that a defective contour and not defective closure of the velopharyngeal sphincter was responsible for a slur of ch or s.

The fact that one observes this functional defect in many normal children who have an extremely irregular contour of teeth strengthens this impression.

Thus one concludes that the anatomic defect of the teeth and the alveolar process is responsible for slurring of ch or s and that defective closure of the velopharyngeal sphincter plays no rôle.

2. Ch or s or both spoken through the nose. There were ten cases in which this defect was present, and in nine some degree of

nasalization was present. In the one case in which no velopharyngeal insufficiency was present there was a small hole anterior to the alveolar process which may have been the cause of the defective articulation.

In two of the cases there were holes in the anterior part of the hard palate. Holes in the anterior part of the hard palate tend to cause an omission of sounds rather than articulation through the nose, as will be demonstrated in the analysis of persons with defects of the hard palate. Therefore, in these two cases the defect was more than likely due to velopharyngeal insufficiency rather than to the holes in the hard palate.

The anatomic defects in this group of cases were as follows: an atrophied premaxilla, one case; malocclusion, one case; orthodontia, two cases; teeth out of line, two cases, and a tooth misplaced posteriorly, one case. In one case there was no anatomic defect of the process but a marked degree of velopharyngeal insufficiency.

As no type of defect of the alveolar process was consistently found in the persons who articulated s or ch through the nose and as velopharyngeal insufficiency was consistently found, the probabilities are that the poor function of the velopharyngeal sphincter was responsible for the phonetic imperfection of articulating ch or s through the nose rather than any anatomic defect of the alveolar process.

3. Omission of s, d, ch or t. There were fifteen cases in which this defect was present. The anatomic defects were as follows: malocclusion, two cases; teeth out of line, four cases; a tooth misplaced posteriorly, one case; teeth abnormally spaced, one case; a small hole anterior to the alveolar process, two cases; overlapping of the medial with the lateral process, one case, and overlapping of the premaxilla by a tooth, one case.

In fourteen cases, or 93 per cent, there was some degree of velopharyngeal insufficiency. In six cases there were holes in the hard palate, besides, the majority being in the anterior part of the hard palate or extending into it.

In one case in which there was no nasalization, the patient had no difficulty with the labial or soft palate consonants, and therefore the probabilities are that the phonetic defect present was due to incorrect habits acquired before operation. The fact that the operation had been performed only six months before this analysis strengthens this impression.

No consistent anatomic defect was found in the alveolar process, whereas velopharyngeal insufficiency was common to all except the one mentioned. This would tend to show that the defect in the alveolar process was not responsible for this phonetic defect but that the inability to close the palate sphincter was the cause.

The question whether or not the omission of these consonants in the six cases in which there were holes in the hard palate was due to these holes or velopharyngeal insufficiency is difficult to determine. Holes in the anterior portion of the hard palate often cause omission of these consonants. These holes were present in only six cases, whereas velopharyngeal insufficiency was present in fourteen. Thus, there were eight cases in which the only anatomic defect was incompetency of the velopharyngeal sphincter.

The impression is that in the omission of the anterior linguopalatal sounds velopharyngeal insufficiency is responsible rather than a defect in the alveolar process unless a hole is present in the hard palate, in which case both anatomic defects are probably responsible. The velopharyngeal defect is possibly the more important of the two.

4. Substitution—d=n, t=d=n, s=n. There were five cases in which this phonetic defect was observed. In all there was some degree of velopharyngeal insufficiency.

As was the case in the articulation of the labial consonants, the consonants requiring less perfect closure of the velopharyngeal sphincter were substituted.

In general, therefore, in persons with defects of the alveolar process velopharyngeal insufficiency plays the major rôle in articulation of the consonants, except the sibilants, in which case the anatomic defect of the alveolar process is more important.

Correlation as Regards Defects of the Hard Palate.—There were twenty-one cases in this group. In fourteen there was no functional defect. In the seven cases in which there were both functional and anatomic defects, four of the patients omitted one or more of the consonants, two spoke the consonants indistinctly and one substituted. All had some degree of nasalization.

Of the twelve cases in which small holes from 1 to 2 mm. in diameter were found in the hard palate phonetic defects were noted in only two. In one case (34) the patient substituted t l for d l. Velopharyngeal insufficiency of 3 degrees undoubtedly was the cause of this defect. In the other case (74), the patient pronounced y indistinctly. Nasalization of 1 degree may have been the cause for this.

In one case (62) a large hole was present in the anterior part of the hard palate, yet in some way the patient articulated all the consonants extremely well. The impression was that the tongue was used to plug the defect.

In one case (91) there were two large holes, one on each side of the palate where lateral incisions had been made. Each hole was 1 by 1 cm. in diameter. The phonetic defect present was a mixture of the more common nasalization plus a quality which is noticed in one who

attempts to talk with hot food in the mouth. This "hot food" quality was also noted in case 63.

In one case (61) a hole measuring 1 by 1 cm. was present at the junction of the hard and the soft palate. All the initial consonants, including k and g, were omitted. Nasalization of 2 degrees was also present, which may have accounted for the omission of the anterior and middle stop position consonants but possibly not for the posterior stop position sounds, k and g.

In two of the four cases in which the patient omitted the consonants there were holes in the anterior part of the palate and in two there were holes in the posterior part of the hard palate.

TABLE 5.—*Analysis of Functional Results in Relation to Anatomic Defects of the Hard Palate*

Case	Anatomic Defect	Functional Defect	Nasalization, Degree
2	Hole, midpalate, 1-2 mm.	0	0
5	Hole, midpalate, 1-2 mm.	0	0
12	Hole, midpalate, 1-2 mm.	0	0
19	Hole, midpalate, 1-2 mm.	0	0
21	Hole, midpalate, 1-2 mm.	0	1
30	Hole, midpalate, 1-2 mm.	0	2
34	Hole, midpalate, 1-2 mm.	t 1 = d 1	3
46	Hole, midpalate, 1-2 mm.	0	0
49	Hole, midpalate, 1-2 mm.	0	0
53	Hole, midpalate, 1-2 mm.	0	0
74	Hole, midpalate, 1-2 mm.	Indistinctly	1
43	Hole, midpalate, 1-2 mm.	0	0
61	Hole, 1 by 1 cm., junction of hard and soft palate	Omits all initials	2
62	Hole, 2 by 1 cm., anterior part of palate	0	1
63	Hole, 3 by 1 cm., anterior part of palate	Omits d 1, 2, 3	1
80	Hole, 1-2 mm., anterior part of palate	Indistinct d 1, 2, 3 th 1, 2, 3	2
91	Two holes, 1 by 1 cm. on each side	0	2 plus "hot food sound"
52	Hole, 1 cm., anterior part of palate; hole, 1 cm., midpalate	t 1, 2, 3 d 1, 2, 3 omitted	1
35	Nasal mucous membrane present in arch	y 1 omitted	1
69	Abnormal scar	0	1
95	Hole, 1-2 mm., junction of hard and soft palate	0	1

In one case (80) in which there was a small hole in the anterior part of the palate, the patient articulated d 1, 2 and 3 and th 1, 2 and 3 indistinctly.

From the statistics alone it is rather hazardous to draw conclusions. A defect of the velopharyngeal sphincter was common to all the cases in which there were phonetic defects, but it was also present in a majority of the cases in which phonetic defects were not present. It is the general impression that when a large hole is present in the hard palate, defective closure of the velopharyngeal sphincter is a minor cause of the phonetic defect in spite of the greater constancy of velopharyngeal insufficiency in all of these cases. Omission of the anterior linguopalatals is apparently the more common phonetic defect of large holes of the palate, especially if the holes are in the anterior part of the hard palate or extend into it. Holes in the lateral part of the hard

palate cause a "hot food" quality to be added to the usual nasalization and cause little or no defect in the articulation of the consonants. Small holes in the middle part of the hard palate cause no articulatory dysfunction.

Correlation as Regards Defects of the Soft Palate.—In this analysis there was a hole in the soft palate in only one case (61). This was large, and in spite of nasalization of 2 degrees it probably was the cause of the omission of the initial k and g.

As for the remainder of the cases, the most important factors seemed to be the motility and length of the soft palate. Consequently, in table 6 the cases in which phonetic defects were present are classified according to the length and motility of the soft palate. The number of cases in which the soft palate was of the same motility and length but in which there were no phonetic defects referable to the soft palate are included for comparison.

The relative importance of length, motility and texture of the soft palate as regards closure of the velopharyngeal sphincter was estimated.

The most common defect in this group was omission of soft palate consonants. A certain degree of velopharyngeal insufficiency, as evidenced by the degree of nasalization, was present in every case except one, and in this one there was merely a slurring of k.

There were phonetic defects referable to the soft palate in nineteen of ninety-seven cases in this group. Three cases were not used in this analysis because two belonged in group III, with clefts of the lip and the alveolar process and normal palates, and there were insufficient data on one.

There can be little doubt that the phonetic dysfunction in these cases was dependent almost entirely on the amount of velopharyngeal insufficiency, granting that there was no hole in the soft palate, as in case 61 in which the hole also played an important part.

Relative Importance of Length, Motility and Texture of the Soft Palate in Closure of the Velopharyngeal Sphincter.—Closure of the velopharyngeal sphincter is dependent on the motility, length and texture of the soft palate. Of the patients with soft palates with motility of 4 degrees, 5.8 per cent (one of seventeen) were defective as far as articulation of soft palate sounds was concerned. Of those with palates with motility of 3 degrees, 16 per cent (seven of forty-two) were defective; of those with palates with motility of 2 degrees, 16.6 per cent (three of fifteen) were defective; of those with palates with motility of 1 degree, 28 per cent (four of fourteen) were defective, and of those with palates with no motility, 50 per cent were defective.

Thus, as the motility decreases, the number of cases of defective articulation increase.

As far as length is concerned, 7 per cent of the patients with long palates had defective articulation of soft palate consonants; 17 per cent of those with median palates had this phonetic defect, and 37 per cent of those with short palates were thus affected.

TABLE 6.—*Analysis of Phonetic Defects in Relation to Anatomic Defects of the Soft Palate*

Case	Motility	Texture*	Length†	Nasalization	Phonetic Defect	Normal Palates with Same Motility and Length But No Phonetic Defects
17	4	F. S.	M	1	k 1, 2, 3 slurred	
	4	F. S.	L	..	.....	
	4	F. S.	S	..	.....	
Total 1						Total 16
14	3	F. S.	L	0	k 1 indistinct	
35	3	F. S.	L	1	k 1; g 1 omitted	
Total 2						Total 12
33	3	F. S.	M	2	k 1, 2, 3 slurred; g 1, 2, 3 indistinct	
21	3	F. S.	M	1	k 1; g	
23	3	F. S.	M	1	k 1, 2, 3 omitted	
90	3	F. S.	M	2	k 1, 2, 3 omitted; g 1 omitted	
Total 4						Total 12
37	3	F. S.	S	1	k 1, 2, 3 omitted	
Total 1						Total 11
60	2	F. S.	M	3	k 1, 2, 3 omitted	
71	2	T. H.	M	1	k 1, 2, 3; g 1, 2, 3 omitted	
Total 2						Total 9
20	2	F. H.	S	2	k 1, 2, 3; g 1, 2, 3 omitted	
Total 1						Total 2
..	2	F. H.	L	..	.....	
Total 0						Total 4
73	1	T. S.	S	3	k 1, 2, 3; g 1, 2, 3 omitted	
77	1	T. S.	S	1	g 1 = y	
86	1	T. H.	S	1	k 1, 2, 3; g 1, 2, 3 omitted	
67	1	F. H.	S	3	k 1; g 1 omitted	
Total 4						Total 1
..	1	T. S.	M	..	.....	
Total 0						Total 6
..	1	T. S.	L	..	.....	
Total 0						Total 3
61	0	T. H.	S	2	k 1; g 1 omitted	
62	0	T. H.	S	1	k 1, 2, 3; g 1, 2, 3 omitted	
78	0	T. H.	S	3	k 1, 2, 3; g 1, 2, 3 omitted	
Total 3						Total 0
..	0	T. H.	L	..	.....	
Total 0						Total 0
..	0	T. H.	M	..	.....	
Total 0						Total 3
56	..	T. S.	L	2	k 1; g 1 omitted	
Total 1						Total 0
19						79

* F. S. indicates full and soft; T. S., thin and soft; F. H., full and hard, and T. H., thin and hard.

† L indicates long; M, median and S, short.

As far as length and motility are concerned, one can make no definite statement concerning their relative importance from the statistics alone, but the general impression is that a person with a short palate with good motility has a better chance of good function of the velopharyngeal sphincter than one with a long palate with no motility. An analysis of the whole group of one hundred cases, including those in the group which have just been analyzed, brings this out also. The results show that 50 per cent of the patients with long palates, 69 per cent of those with median palates and 54 per cent of those with short palates had nasalization, while 55 per cent of those with palates with motility of 4 degrees, 62 per cent of those with palates with motility of 3 degrees, 64 per cent of those with palates with motility of 2 degrees, 81 per cent of those with palates with motility of 1 degree and 100 per cent of those with palates with no motility had some nasalization.

Motility seems to be the more consistent as regards the presence of velopharyngeal insufficiency.

As regards the consistency of the soft palate and its effect on articulation, it can be concluded that while it has no effect per se it has an effect secondarily by its relation to motility. The average motility of the full, soft palates is 3; that of the thin, soft palates is 2; that of the full, hard palates is 1.5, and that of the thin, hard ones is 1.1.

Motility is therefore of more importance than length, although the latter is of marked importance for competent closure of the velopharyngeal sphincter. As far as can be determined, little emphasis has been given to possible injury to the nerve supply of the palatal muscles by the different types of operations. No description of the course and distribution of the nerves supplying the tensor veli palatini or the levator veli palatini muscle has been found. It seems of great importance as far as preservation of motility is concerned to demonstrate their course if possible and to ascertain the danger of injuring them in the different types of operation for repair of the soft palate.

Superior Constrictor Muscle.—That the superior constrictor muscle elements are well developed in cases of cleft palate is evident. In this series 88 per cent were classed as well developed. Many of these were apparently hypertrophied, although no definite comparison was made with the normal. The general impression is, however, that the superior constrictor muscle elements are better developed in cleft than in normal palates.

The fact that all repaired cleft palates are as a rule shorter and less mobile than normal palates and yet adequate closure is obtained frequently tends to suggest the importance of this hypertrophy of the superior constrictor muscle for speech.

Importance of the Uvula.—Sixty-one per cent of the patients having long uvulas had some nasalization; 55 per cent of those with median length uvulas were thus affected; 71 per cent of those with short uvulas had nasalization; 78 per cent of those with no uvula were thus afflicted, and 56 per cent of those with bifid uvulas had nasalization.

As there is little consistency in these results, this would tend to show that the uvula is of negligible importance in closure of the velopharyngeal sphincter.

Effect of Adenoidectomy and Tonsillectomy.—That adenoidectomy and tonsillectomy considerably enhance the ability to speak well has often been noted.

One patient (in case 6) was observed before adenoidectomy and tonsillectomy and was seen subsequently one week and again one month postoperatively, and a report from the mother was received three months after operation.

Before operation the result had been classed as excellent. There was no phonetic defect, and the palate had a motility of 3 degrees and was of median length. One week postoperatively the motility and length of the palate were the same, but nasalization of 2 degrees was present. The articulation of the consonants was still good, but the nasalization had changed the quality of the speech markedly. The result of the functional test was poor. One month after operation there was less nasalization (1 degree), less difficulty with the functional test was found and the quality of the voice was improving. Three months after operation, the mother reported "steady improvement" in the speech.

It is impossible to draw conclusions from one case, but it seems reasonable to concede that if a mobile palate of good length is present, tonsillectomy and adenoidectomy can be performed without danger of permanent damage to speech. One feels, however, that if the palate is inadequate as to length and motility in the first place, adenoidectomy or tonsillectomy should not be performed.

Value of Speech Training.—Fifty-six per cent of the patients in this series have had no speech training. Twenty per cent have had only occasional corrections by the family. Six per cent have had more persistent corrections by the family. Eight per cent have had occasional lessons in speech. Fourteen per cent have had persistent teaching by well trained teachers.

Fifty-one per cent of the fifty-six who had had no training were classed as excellent as regards their speech, while 28 per cent had good speech, 20 per cent had fair speech and not one had bad speech.

At the other extreme, only 36 per cent of the fourteen who had had adequate training had excellent speech, 57 per cent had good speech, 7 per cent had fair and none had bad speech.

At first glance, these results would condemn speech training as useless, but a study of the anatomic structures of the two groups brings out the fact that a greater percentage of the patients who have had no training have good anatomic structures, whereas in the group which has had training there is a greater percentage of anatomic defects.

Moreover, as a rule, only the patients with the poorest functional results have been referred for speech training.

There have been several patients who had had speech training, and although they still have a marked degree of nasalization the articulation of the consonants at the stop position is excellent.

Speech training is most important in perfecting correct articulation. It also assists in developing closure of the velopharyngeal sphincter, but with inadequate anatomic material it cannot possibly do this.

Ultimate Speech Result.—Of the one hundred cases presented, the ultimate speech in 42 per cent was classed as excellent, in 43 per cent as good, in 14 per cent as fair and in 1 per cent as bad or unintelligible.

CONCLUSIONS

1. Good anatomic results do not necessarily mean good functional results.

2. Imperfect closure of the velopharyngeal sphincter is by far the most important anatomic element in ultimate speech results.

3. Velopharyngeal insufficiency is the most important factor in the omission, substitution and disarticulation of most of the consonants. The slurring of s and ch is an exception.

4. Velopharyngeal insufficiency is the cause of the nasalization and therefore the disturbed quality of cleft palate speech. There is a possibility that scar tissue and taut mucous membrane play a rôle in the poor quality by disturbing the vibratory capacity of the cavities of the upper respiratory passage.

5. Abnormal contour of the teeth causes slurring of s and ch.

6. Holes just posterior to the alveolar process in the anterior part of the hard palate have a tendency to cause omission of the labiodental consonants.

7. Small holes in the hard palate cause no phonetic defect.

8. Large holes in the middle portion of the hard palate tend to cause a "hot food" quality to speech rather than a disturbance of articulation.

9. Holes in the lateral part of the hard palate tend to cause a "hot food" quality to speech.

10. Holes in the soft palate tend to cause omission of the posterior linguopalatal consonants.

11. The motility of a palate appears to be more important than length as regards closure of the velopharyngeal sphincter.

12. The superior constrictor muscle elements are important in closure of the velopharyngeal sphincter and are usually hypertrophied.

13. The uvula may be of importance in closure of the velopharyngeal sphincter, but the analysis does not indicate this.

14. A person with a cleft palate usually has distorted nasal cavities, and occasionally the inferior turbinate bones are enormously dilated.

15. Adenoidectomy and tonsillectomy are indicated if the motility and length of a palate are good.

16. Speech education is of greatest value in correcting articulation.

17. No amount of education will entirely overcome marked velopharyngeal insufficiency, although it is of definite value in lesser degrees of insufficiency.

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PYOGENIC OSTEOMYELITIS OF THE PELVIS

ANALYSIS AND DISCUSSION OF NINETY CASES

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This study demonstrates the essential similarity of pelvic osteomyelitis to the more common osteomyelitic manifestations in the long bones. However, pelvic localization is distinguished by its anatomic situation, as defined by the peculiar developmental configuration of the innominate bone and its relation to the adjacent fascial spaces, viscera and the hip joint. These features anticipate the differential diagnostic problems encountered. It is characteristic of the disease that the primary lesion is overlooked or subordinated to the predominant simulative signs and symptoms or to the more extensive secondary parosteal purulent infiltrations. Therefore, pyogenic osteomyelitis of the pelvis continues to challenge diagnosis and treatment during its earlier, more favorable stages, although it is a relatively common devastating disease.

In a series of 1,496 cases of pyogenic osteomyelitis, there were 90, or 6 per cent, in which the focus of infection was in the pelvis. This lesion was third in the order of relative frequency. The ratio (table 1) of males to females in the group of 90 cases was approximately 2:1, except in the group in which the infection was in the sacro-iliac region. The age range was from 2 to 63 years, with an average age of 16 years. Pelvic osteomyelitis is primarily a disease of the growing period. The majority of cases (about 60 per cent) occurred in the second decade. Because of the relation of the sacro-iliac region to spinal foci, there is a relative increase in the later decades.

PATHOGENESIS (TABLE 2)

The apparent port of bacterial entry could be determined in 20 instances. In 17 it was a topical infection and in 3 an acute infectious disease. Boils were the predominant localized infection of the skin. The relationship between such ports of entry and the osteomyelitic process is not always clearcut. Adequate treatment of all topical focal infections, as well as of acute infectious diseases, is an important prophylactic measure. Antecedent trauma was recorded in 35 cases. In most of these the onset of symptoms followed direct or indirect injury,

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sometimes dramatically so. The crests of the ilium are not well protected by the soft parts. The sacro-iliac joints are notoriously prone to indirect strains. The ischium and the pubis are readily injured by falls on the buttocks and especially by straddling. The relationship between antecedent trauma and pelvic osteomyelitis substantiates the time-honored assumption of its etiologic importance.

Pelvic osteomyelitis may be classified as a direct or a hematogenous infection. The former follows gunshot wounds, operations and compound fractures or is due to extension from a neighboring suppurative process and infected decubiti. There were 23 (about 26 per cent) cases of direct infection. In 2 cases sacro-iliac osteomyelitis followed arthrodesis, in 1 it was due to extension from an iliac osteomyelitis and in 7 it was associated so closely with disease of the lumbosacral region as

TABLE 1.—*Sex and Age Incidence in Ninety Cases of Pyogenic Osteomyelitis of the Pelvis*

	Ilium	Sacro-Iliac Joint	Ischium	Pubis	Total
Sex:					
Males	20	22	14	6	62 (68%)
Females	11	7	7	3	28 (32%)
Age, years:					
Youngest	4	5	2	4	3
Oldest	63	50	33	59	51
Average	16	21	11	16	16
Decades:					
1	5	2	9	3	19 (21.11%)
2	21	19	10	3	53 (58.89%)
3	1	1	1	1	4
4	2	1	1	1	5
5	1	5	0	0	6
6	0	1	0	1	2
7	1	0	0	0	1

to preclude initial localization.. In 4 cases iliac osteomyelitis was due to extension from the initial lesion of the hip, and in 1 it resulted from an infected compound fracture. In the 4 cases of ischiac osteomyelitis the condition originated from primary foci of the hips. In 1 case pubic osteomyelitis occurred after a simple fracture, which was operated on and fixed by wire; in 2 it followed pelvic fracture and was associated with genito-urinary complications, and in the fourth it was due to a dissecting superficial abscess of the groin.

Sixty-seven (74 per cent) of the cases of pelvic osteomyelitis were of the hematogenous form; the lesions in 55 were initial, and in 12, metastatic after the establishment of a bony focus elsewhere. On the basis of the predilection of pyogenic osteomyelitis for growth zones, a diphasic clinical syndrome may be observed as a general rule, corresponding to the two periods of bony development. The first period extends from infancy to puberty, at the end of which period the three bones composing the acetabulum fuse. The second period commences

with the ossification of the acetabulum and the appearance of the marginal epiphyses and extends to the time of fusion of the marginal epiphyses, at about 25 years of age. The first period is characterized by a more diffuse lesion, almost invariably occurring at the border of the acetabulum. The second period localizes about the marginal epiphyses. In the sacro-iliac region the initial process begins most commonly on the sacral side but may originate in the ilium or the joint proper. Very soon, however, the entire joint is involved.

Staphylococcus was recovered in the great majority of instances. Sterile cultures are also significant, since the organism may be in the bone and not free in the exudate. Sterile cultures always indicate a most careful laboratory search to rule out tuberculosis, a malignant process or some unusual bacterial invader. The mixed forms are secondary invaders for the most part. Careful initial blood and local cultural studies reveal not infrequently the presence of several organisms, which

TABLE 2.—*Pathogenic Classification of Cases of Pelvic Osteomyelitis* *

	Ilium	Sacro-Iliac Joint	Ischium	Pubis	Total
Direct infection	5	10	4	4	23 (25.55%)
Hematogenous infection					
Primary lesion	20	16	15	4	55 (61.11%)
Metastatic lesion	6	3	2	1	12 (13.33%)
Infectious agent					
Staphylococci	12	14	12	4	42
Streptococci	3	2	0	0	5
Mixed infection	2	2	1	0	5
Sterile culture	1	4	0	1	6
Positive blood culture	4	2	4	0	10

* Possible ports of bacterial entry and antecedent trauma play an important rôle in the apparent pathogenesis in this series. The latter was recorded in 35 instances of the disease in the following sites: sacro-iliac joint, 12; ilium, 8; ischium, 11, and pubis, 4. There were 17 recorded instances of previous topical infections, which occurred as follows: sacro-iliac joint, 6; ilium, 5; ischium, 5, and pubis, 1. There were 3 instances of previous acute infectious diseases: sacro-iliac joint, 1, and ilium, 2.

are apparently simultaneous primary bacterial invaders. Most frequently there is recovered a combination of staphylococci and streptococci. A positive blood culture yielding staphylococci or streptococci is of considerable differential diagnostic importance.

PATHOLOGIC ANATOMY (TABLE 3)

There were 4 cases in which the lesions were bilateral, all of which proved to be fatal; in 3 the sacro-iliac joints and in 1 the ischiums were involved, making a total of 94 foci. The cases of bilateral sacro-iliac osteomyelitis were associated with involvement of the lumbosacral region, and the cases of ischiac osteomyelitis, with extensive hemipelvic disease. Pelvic localizations are all more or less diffuse, owing to the peculiar bony configuration and the tendency to chronicity, especially after obliteration of cartilaginous barriers. Seventy per cent were associated with other segmental foci, chiefly by extension. The hip joint is the most frequent site of an associated focus.

The most striking pathologic feature of pelvic osteomyelitis is the relatively "dry" bony reaction as a subacute or chronic fibrous type of osteomyelitis. Conversely, extensive suppuration of the soft tissue is characteristic. The fibrous reaction is partly due to collateral ischemia. The initial site of involvement usually reveals a more active inflammatory reaction. Sequestration is not as uncommon as the literature would indicate. The preponderance of cancellous bone, negligible marrow cavity and excellent circulation is inducive to caries rather than to necrosis. In the sacro-iliac region sequestrums are small and usually situated antero-inferiorly. The changes in the joints are the same as those of infectious arthritis elsewhere. The thin cortical bone of the iliac wing leads to early perforation and variable-sized sequestrums, especially on its pelvic surface. Consequently, it is common to observe at operation the formation of large irregular cisterns on the pelvic surface of the bone, which are filled with pus, sequestrums and débris

TABLE 3.—*Pathologic Anatomy in Cases of Pelvic Osteomyelitis*

	Ilium	Sacro-Iliac Joint	Ischium	Pubis	Total
Unilateral osteomyelitis	31	26	20	9	86
Bilateral osteomyelitis	0	3	1	0	4
Extensive lesion	17	20	13	3	53 (58.88%)
Localized lesion	14	9	8	6	37 (41.12%)
Associated extension Pelvic lesions					
Hip	13	5	7	1	26 (44%)
Ilium	..	7	3	..	10
Ischium	3	..	..	1	4
Pubis	1	..	4	..	5
Lumbosacral portion of spine	..	8	..	..	8
Sacro-iliac joint	6	..	..	..	6
	23	20	14	2	59 (70%)

(fig. 12). Occasionally the entire wing is converted into a variegated necrotic mass. At operation in the later stages of the disease the ischium lies in a soggy bed and can be lifted out *en masse*.

A striking feature of the later stages is the amount and irregularity of new bone formation. The periosteum is very active in cases of pelvic osteomyelitis, and the tendency toward regeneration—even after total resection—is almost certain in the young. This regenerative irregularity often adds to the operative difficulties encountered. The power of the periosteum may be weakened or inhibited entirely in older persons or because of constitutional inferiority, initial thrombosis and death of the periosteum or intense parosteal infection. Necrosis and intense edema of the surrounding soft parts may predominate the situation. Even in the early stages the histologic tendency toward thickening and eburnation of the bony lamellae is conspicuous.

COMPLICATIONS (TABLE 4)

The complications of pelvic osteomyelitis especially reflect the clinical life history. The dovetailedness of these phenomena reveal an apparent

harmony, imperceptible from any other perspective. It is now evident that most complications are of surgical interest, since the vast majority are dependent on the local skeletal lesion. Table 7 indicates this clearly.

Suppuration was observed in over 83 per cent of this series and largely determined the course of the disease. These abscesses tend to gravitate and accumulate in the anatomically related fascial spaces of the thighs, the lower part of the back and the gluteal regions, which are easily invaded. Such purulent collections predominate the surgical situation not alone because of their complexity and bulk but because their shaggy infiltrated walls become in themselves formidable sources of local and systemic infection.

The possible pathways taken by pus originating in the sacro-iliac joint after it has perforated anteriorly illustrate the ramifying tendencies of all pelvic lesions. Pus may follow along the iliopsoas muscle to the inner aspect of the thigh or along the pectineus muscle posteriorly; it may enter the hip joint via a bursa found between the iliopsoas tendon and the anterior part of the joint, or it may burrow posteriorly along the obturator internus muscle or along the pyriformis muscle as a low gluteal abscess or upward from the iliac fossa into the lumbar region. Abscesses arising from iliac foci are of special interest because of their relation to the inner surface of the bone, which is divided by the iliopectineal line into an upper, expanded, portion and a lower, smaller, part. The iliac fossa above and anterior is smooth by sloping, which explains the great prevalence of abscess formation in this region and its tendency to point anteriorly in Scarpa's triangle; since pus is blocked from passing farther into the true pelvis by the structures attached to the iliopectineal line, gravity forces it forward rather than backward. Infected compound fractures of the pubic bones are especially interesting because of the relationship of the bladder and the posterior portion of the urethra. An extensive abscess in the space of Retzius was observed in 1 case in which the pyogenic infection was associated with traumatic rupture of the posterior portion of the urethra. Suprapubic cystotomy and perineal urethrotomy were performed, and the patient died after a stormy course of three weeks' duration.

CLINICAL ANALYSIS (TABLE 5)

In a general way the onset of the disease determines whether the local or the systemic reaction will dominate the initial clinical stages. The disease becomes chronic in practically all survivors, as shown by the average duration of symptoms in this series. The paucity of recorded clinical dates emphasizes the unsuspected nature of the disease in many of these cases. There are, however, sufficient facts obtained to indicate the general clinical picture. It should also be remembered that rather

extensive destructive lesions of a low grade nature may present few or no symptomatic clues. In retrospect, it is evident that a knowledge of the disease is of primary importance for a reasonably early diagnosis.

DIAGNOSIS

The chief differential diagnostic interest centers about the hip joint. However, this also involves a discussion of a wide variety of other

TABLE 4.—*Complications in Cases of Pelvic Osteomyelitis*

	Ilium	Sacro-Iliac Joint	Ischium	Pubis	Total
Suppuration	27	21	18	9	75 (83.33%)
Pelvic	15	10	2	1	28 (37.33%)
External	7	4	11	3	25 (33.33%)
Thigh	5	7	5	5	22 (29.44%)
Flare-up	13	17	7	3	40 (44.44%)
General sepsis	12	15	5	5	37 (41.11%)
Spontaneous sinus	9	11	7	2	29 (32.22%)
Deformity	15	4	6	1	26 (28.88%)
Skeletal metastasis	9	6	7	1	23 (25.55%)
Sequestrum formation	11	4	4	2	21 (23.33%)
Vascular complications	2	2	..	..	4
Decubiti	1	2	..	..	3
Amyloidosis	..	2	..	..	2
Rectal perforation	1	..	1	..	2
Peritonitis	1	..	..	..	1

TABLE 5.—*Clinical Analysis of Cases of Pelvic Osteomyelitis*

	Ilium	Sacro-Iliac Joint	Ischium	Pubis	Total
Onset					
Acute	13	11	9	5	38
Subacute	7	7	5	4	23
Chronic	6	3	2	..	11
Duration, months					
Shortest	0.15	0.3	0.1	0.1	0.16
Longest	676	84	276	132	292
Average	47	27	34	35	33
Symptoms					
Subjective					
Pain localized	7	10	4	4	25
Pain referred	9	10	6	2	27
Objective					
Local tenderness	8	10	10	3	31
Muscle spasm	3	3	..	1	7
Loss of weight	9	8	3	4	24
Superficial abscess	8	3	6	3	20
Pelvic abscess	5	6	..	..	11
Edema and infiltration	5	6	6	2	19
Deformity of hip	10	13	7	1	31

extrapelvic lesions, such as inflammation of the lower part of the abdomen and pelvis, spondylitis of the lumbar and lower part of the dorsal region, subgluteal bursitis, ischiorectal abscess, isolated abscess of the iliopsoas muscle and bursitis, as well as malignant lesions and tuberculosis. Ruling out actual involvement of the hip joint offers the greatest clinical obstacle to a reasonably early diagnosis, since a sympathetic juxta-articular effusion occurs so frequently. It should be remembered

in this connection that the joint may be the primary seat of infection, be affected simultaneously or included in the process by extension secondarily. Marked symptoms referable to the hip joint, especially when accompanied by radiating pain along the internal obturator nerve, are almost pathognomonic of relative or absolute participation of the hip joint in the disease.

Primary disease of the hip joint is early associated with more or less of a position of instability of the joint, mainly flexion, abduction and external rotation. The flexed position may be combined with adduction and internal rotation. This position is conditional on the relationship of the initial localization of the infection to the anatomic quadrants of the acetabulum, the head of the femur or the pelvifemoral musculature. Thus the resultant subsequent position of the hip is maintained by a specifically activated protective muscle spasm pattern characterized by relative or absolute spasm. However, some degree of motion may be elicited before the joint cartilage is involved, or in the more chronic lesion due to the capsular and ligamentous relaxation incident to a prolonged state of hydro-arthrotic distention. When the disease is in the pelvis, the attitude of the hip and the limitations of joint motion are less significant. Extra-articular conditions will allow a considerable range of motion in the joint in the planes not controlled by those pelvifemoral muscles arising or inserting at sites of bony involvement. Therefore, the attitude of the hip is now sympathetic and relatively atypical. When the focus is in the ilium, the hip is characteristically in flexion and abduction. Flexion predominates when the focus is in the sacro-iliac joint. When the infection is localized in the ischium, external rotation of the femur is present. If the disease is in the pubis, there follow flexion, adduction and internal rotation as a rule.

Aspiration of the hip joint and surrounding structures is the most important single factor in the clinical evaluation of suppurative localizations. This must be persisted in intelligently and may be performed with the patient under general anesthesia. The gross and laboratory examinations of the material withdrawn yield the key to the situation.

Special emphasis should be given to the infection in the sacro-iliac region, since it involves the syndrome of pain low in the back. It may be impossible to make a differential etiologic diagnosis without exploration and biopsy. Even so, the laboratory tests were misleading in several cases in that the exudate yielded a positive reaction when injected into a guinea-pig, but the reaction was not substantiated by subsequent autopsy examination or pathologic studies of sufficient material removed at operation. Disease of the sacro-iliac joint often simulates disease of the hip joint. On examination the motions of the hip will be free, except the extremes of flexion and extension, because the former puts tension on the hamstring muscles, which is transmitted to the sacro-

iliac joint, and in sacro-iliac involvement the latter is restricted by associated spasms of the iliopsoas muscle. It is most difficult to differentiate sacro-iliac osteomyelitis from sacro-iliac tuberculosis. Ombredanne considered the latter to be rare in infancy, and Wilmath stated that it is exceptional before the age of 20. In children the tuberculous process of the os ilium originates at some distance from the bone as osteomyelitis. It is more rapid in the adult and is primarily an involvement of the joint or osteo-arthritis. The initial reaction may, however, be ushered in by a septic fever or the appearance of a tuberculous focus in other places. In the adult type of sacro-iliac tuberculosis in the early stages there are five symptoms of value: sacroradiculitis, painful limp, sacro-iliac diastasis (noted roentgenographically) and upward displacement of the pubic bone and, finally, localized surface and rectal tenderness, elicited digitally.

Abdominal and pelvic inflammations are simulated chiefly by localizations of the disease in the sacro-iliac joint and in the ilium, especially in the latter. In the acute stages the profound general reaction and bacteremia point toward pyogenic skeletal disease. Careful observation of spontaneous and of provoked pain is a great aid in the diagnosis. Spasm of the abdominal muscles, when present, confines itself to the iliac crest and soon takes on the character of induration and even of edema. This tenderness even very early may be outlined in the posterior portion of the ilium along the crest or even may simulate a perinephritic abscess. Tenderness is common in the groin but cannot be accurately limited. When perforation occurs, the temperature and the general condition are improved. Some pain persists, however, and soon a purulent collection in the pelvis is clinically evident. In acute pubic lesions one has to distinguish between phlegmonous inflammation in the space of Retzius, suppurative arthritis, psoasitis and myositis of the rectus abdominalis muscle. Ischiac lesions offer no particular difficulties if the examiner thinks of the possibility.

ROENTGENOGRAPHIC EXAMINATION

The roentgenogram is indispensable for diagnosis and proper surgical evaluation, particularly in the subacute and chronic stages of the disease. The entire pelvis and the lumbar portion of the spine must be carefully checked after the patient has been properly prepared. Several pronounced lesions of the iliac wing were missed early in the series because of failure to observe this simple detail. Lateral views of the iliac bones and hips are helpful, since localized lesions of the ilium can be visualized even early. Although the general operative risk determines the surgical indication, no operative procedure can be intelligently planned without a complete preliminary roentgenographic study of the

situation. The changes in the soft tissues are of some significance in the early stages of acute inflammatory conditions and are observed as capsular and other fascial and muscular distortions and infiltrations. In cases of ischiac lesions the obturator foramen may be clouded early because of abscess formation. The hip joints must be extended if possible to avoid superimposed shadows from the tissues of the thighs. The essential changes depicted by the roentgenogram are osteoporosis, sequestration and bone production. Osteoporosis is the earliest sign in the acute stage. Its delay is due to the fact that the calcium salts early remain at the site of involvement in an attempt to limit the pathologic process. It appears soonest in the more primitive cancellous portions of the bones in which calcium is not so bound up as in the more highly organized haversian architecture prevalent in cortical bone. Because osteoporotic changes sometimes persist, these lesions simulate tuberculosis and even malignant processes. Except in the pubis and the sacro-iliac region, the regenerative features are characterized by great thickening and irregularity. In the chronic phases there is left a mass of irregular sclerotic thickened periosteal bone with little or no architectural detail that can be made out. The value of injection of a sinus with a radiopaque substance cannot be overemphasized as an aid to diagnosis.

END-RESULTS (TABLES 6 AND 7)

The inadequacy of treatment previous to hospitalization reflects the general attitude of the medical profession toward this disease. These lesions have always been considered as hopeless and attended by a grave prognosis. Of the 29 cases of sacro-iliac lesions, the superficial abscess was incised in 7 and aspirated in 2. Of the 21 cases of ischiac osteomyelitis, incision was performed in 5 and curettement in 1. Of the 21 cases of iliac lesions, curettement was performed in 4 and simple incision in 4. The pubic lesions were not treated. The treatment in the hospital also indicates an absence of definite principles in the earlier cases. On the whole, the best results were obtained after the need for more adequate treatment was recognized.

Of the 18 cases of sacro-iliac osteomyelitis in which the bone was attacked, curettement was done posteriorly in 5, and the Smith-Petersen technic was used in 5, total resection in 3 and partial resection in 5 (1 bilaterally). The Smith-Petersen technic is adequate for circumscribed lesions, especially when reenforced by Orr's postoperative routine. In 1 case this procedure was followed by total resection at a later date. In the second case two further operations were necessary. In 2 the lesions were not suppurative, and in the fifth a subgluteal abscess developed one month after operation. In 7 cases simple incision and drainage were employed, the only favorable result being obtained

by the Orr method. There was no operative mortality in the group in which resection was done. Three patients died subsequently from complications already present at the time of operation. In 5 cases healing occurred in from six months to one year.

In the group of patients with iliac osteomyelitis there were 2 adolescents (1918 and 1927) who were treated by roentgen irradiation, with apparent cessation of the clinical symptoms. Five of the patients

TABLE 6.—*Treatment and End-Results in Cases of Pelvic Osteomyelitis* *

Type of Treatment	End-Result	Ilium	Sacro-Iliac Region	Ischium	Pubis	Total
Radical	Healed	7	13	4	2	26
	Died	1	3	2	1	7
	Unknown	2	2	2	0	6
Incision	Healed	1	1	2	1	5
	Died	3	1	1	0	5
	Unknown	9	3	3	3	18
Conservative	Healed	4	1	0	0	5
	Died	0	0	1	1	2
	Unknown	4	5	6	1	16

* This table classifies the treatment as a whole. Radical treatment indicates that the affected bone was attacked in one way or another. The affected bone was not attacked at all in the cases included under simple incision. Conservative treatment includes roentgen irradiation, the use of casts or other mechanical methods, simple hospitalization or no active treatment at all. The mortality as regards the cases as a whole is: radical treatment, 18 per cent; incision, 18 per cent, and conservative treatment, 9 per cent. However, on the basis of the cases in which the end-results were known the mortality is as follows: radical treatment, 21 per cent; incision, 50 per cent, and conservative treatment, 40 per cent.

TABLE 7.—*End-Results in Unselected Cases of Pelvic Osteomyelitis in which Resection Was Performed*

Localization	Operation	Number of Cases	Healed	Died	Mortality
Sacro-iliac joint	Partial resection	5	3	2	40%
	Total resection	3	2	1	33.33%
Ilium	Partial resection	4	3	1	25%
	Total resection	1	1	0	0
Ischium	Total resection	4	4	0	0
		17	13 (76.48%)	4	23.52%

were not treated for one reason or another. Three were treated by sequestrectomy without success. Thirteen were treated by simple incision and drainage; healing occurred in 1, and 3 died. Of the more radical procedures, total resection was performed once, partial resection four times, saucerations several times and trephination once. In the case in which trephination was done over 50 metastatic foci subsequently developed. There was only 1 death in this group.

Simple incision and drainage were performed in 6 cases of ischiac osteomyelitis, with cures from well circumscribed foci in 2. The disease was bilateral in 1 of the remaining 4 cases, and it extended to the hip

in 1 and to the ilium in the other. In the fourth case the condition proved fatal. Death also occurred in the 2 cases in which sequestrectomy was performed and in 1 case in which a simple incision was done. In another case a mistaken diagnosis of disease of the hip joint was made, and operation was performed, with fatal results. The radical operations (resection) were performed in the acute and subacute stages of the disease with sound healing within from three to six months.

In the 5 cases of direct infection of the pubic bone, simple incision and drainage were employed. Autopsy in the fatal cases revealed a huge abscess in the space of Retzius, which extended laterally into the iliac fossae. The metastatic focus in the cases of hematogenous infection drained spontaneously. Sequestrectomy was performed on a boy two weeks after onset, with healing in four months. Simple incision on a 4 year old girl, three weeks after an acute onset, ended in extension of the lesion and death. A youth aged 17 years was treated by a cast, with subsequent invasion of the anterior compartment of the thigh. The most gratifying and convincing result followed prompt adequate treatment in a case of acute involvement in which the abscess and involved bone were treated by the Orr method.

TREATMENT

The need for radical treatment was recognized and urged by the earliest writers on iliac lesions. Perhaps their very zeal delayed the acceptance of these principles. An extended experience with pyogenic osteomyelitis supports this fundamental therapeutic truth relative to established foci of the disease. One should remember that the lesion may be nonsuppurative and that spontaneous healing may occur. However, pelvic osteomyelitis is essentially a most devastating disease which demands bold operative measures. The key to the individual situation is revealed by the complications of the disease in general. The vast majority of these depend primarily on the skeletal lesion and only secondarily on any existing systemic reaction. Therapy now resolves itself into the primary and secondary control of the disease.

Primary control of the disease involves the initial systemic infection and the determination of the local suppurative focus. These must be considered in the order of their appearance. Henceforth the most aggressive measures continue to be directed against whichever predominates the clinical situation. Until contradictory knowledge affecting the relationship between the port of bacterial entry, the systemic infection and the local lesion is attained, adequate drainage is indicated when localization is clinically established. Pyogenic osteomyelitis anywhere is not a surgical problem until this occurs. The successful termination of the acute stages of the disease depends on the surgeon's experience

with the disease. Therefore, treatment at the onset must be directed against the systemic situation by the several means at one's command —dextrose solution intravenously, small repeated transfusions, sedatives, splinting, traction or casts—and one should ever be on the watch for localizing skeletal signs and symptoms. Perhaps early diagnosis and immediate bony drainage have been overemphasized at the expense of a careful evaluation of the surgical risk. The fault rests with the individual surgeon. Poor preoperative surgical judgment reflects unfairly on and discredits sound axiomatic principles of drainage. However, the conservative postoperative technic of Orr permits more extensive surgical treatment in the early stages than would otherwise be feasible in pelvic localizations of the disease.

As a rule the signs of localization in the soft tissue appear within a few days of the onset of symptoms. Simple incision and drainage will suffice until more definite pathologic skeletal delineation is manifest. By this time the operative risk and the operability of the lesion will determine all further operative details. Wide excision of affected bone is the ideal to be attained. Several factors are effective indicators of improvement of the patient's operative risk and of definite establishment of localization of the disease: a decrease in the initial rapidity of the sedimentation rate, intermittent fever after an initially sustained level of temperature, associated with a lowered pulse rate, a fair cellular blood level and even diminished apprehensiveness and pain.

Secondary control of the disease is based on anticipation of pathologic extension, sequestrum, secondary infection, persistent sinus and flare-ups and above all on locomotor disturbances. The patient now definitely becomes a veritable battleground between the conscientious surgeon and complications, which are practically all preventable or amenable to treatment. Extension of the disease involves chiefly the adjacent joint or joints and the fascial spaces and their contents. Sinus formation enhances secondary infection or even malignant change. Locomotor disturbances are caused principally by deformities of mechanical origin. All of these can also be most uniformly prevented by observing Orr's second great principle, that of adequate rest of the parts.

Pelvic osteomyelitis adapts itself to radical treatment in spite of the apparent complexity of the anatomic situation. The peculiar anatomic structure of the bony pelvis permits a diffusion of functional stresses for its weight-transmitting function through its posterior and anterior arches. The complexity of the soft tissue elements of the pelvis is compensated for by the ready subperiosteal surgical exposure of the bony segments.

The irregular structure of the innominate bone is dissipated when it is regarded as a figure 8 twisted on itself. The acetabulum is there-

fore situated at the isthmus. The upper two-fifths forms the ilium; the ischium comprises the lower and posterior two-fifths, and the pubis, the remaining one-fifth. The fairly even distribution of the parts is not generally appreciated. The thicker parts of the iliac bone consist of cancellous tissue enclosed within two layers of compact tissue, which are lined by periosteum. The thinner parts of the bottom of the acetabulum and the center of the iliac fossa are transparently thin and composed entirely of compact tissue. The inner surface of the ilium is of interest in relation to pyogenic infection. In general, the ischium is the posterior and inferior portions of the lower expanded part of the innominate bone and helps to form the posterior boundary of the obturator foramen. The body is the lower two fifths of the acetabulum and spine of the ischium and is limited below by its neck. The posterior border of the neck is the lesser sacrosciatic notch. The tuberosity of the ischium continues down from the neck to the ascending ramus and forms the rocker of a rocking chair. It is triangular in cross-section. The ascending ramus is a thin plate of bone going up to meet the descending ramus of the pubis. These two pubic bones complete the pelvic girdle and consist of a body and a descending and an ascending ramus. The descending ramus meets the ischium and completes the boundary of the obturator foramen. The ascending ramus goes laterally from the body and upward to end in the acetabulum. The body is the portion at the symphysis and has a quadrilateral form. Its upper border is thicker than the lower.

The sacro-iliac joint is a true joint cavity and has a synovial lining, which is supported by a capsule. The joint surfaces are ordinarily smooth, save for sufficient roughening to lock the joint surfaces. The lumbosacral cord lies directly over the lower third of the sacro-iliac joint. In this region nothing except the joint capsule intervenes between the nerve trunk and the joint space. The proximity of the superior gluteal vessel to the inferior margin of the greater sciatic notch offers a real danger during resection of the sacro-iliac joint and ilium. Enormous superior gluteal arteries, often the size of a man's thumb, have been found in cadavers. The vessels are separated from the ilium only by the periosteum. They are about 1 inch (2.5 cm.) long and have a tendency to retract into the pelvis when severed. Other structures in close proximity to the anterior portion of the sacro-iliac joint in the pelvis that are of surgical importance are the great pelvic vessels, the ureters, the lumbosacral plexus and the rectum. The rectum forms at the third sacral segment and accommodates itself to the hollow of the sacrum and the coccyx. All of these are more or less protected by the iliopsoas structures, which are further thickened by inflammatory edema and infiltration when the sacro-iliac joint is involved by osteomyelitis.

The sacro-iliac joint and adjacent structures are attacked by the Bardenheuer-Picque technic.

In resection of the ilium a Smith-Petersen incision permits the entire external soft tissue flap to be stripped down to the margin of the acetabulum and is followed by anterior deep dissection to expose the anterior portion of the ilium. Posteriorly, the exposure continues to the greater sciatic notch. Next a similar subperiosteal exposure is made of the internal surface of the ilium. At this stage it is remarkable to note how the abdominal contents fall away to complete the exposure of the wing of the ilium down to the arcuate line of the ilium. In cases of chronic involvement subperiosteal dissection will be somewhat difficult because of scarring due to perforation and the formation of an involucrum. One may then remove the ilium as indicated or *en masse* to the supracotyloid region.

My technic for resection of the ischium employs a posterior vertical or transverse gluteal approach, because it is adequate, is farther from the anal canal, furnishes a thick soft tissue pad for subsequent healing and does not require any unusual position of the patient. An incision of from 4 to 6 inches (10 to 15 cm.) long is made over the lateral margin of the tuberosity of the ischium. The dissection continues to the tuberosity. The lowermost fibers of the gluteus maximus fibers are encountered and may be retracted upward or incised near their lower origin. Next the sciatic nerve is located lateral to the tuberosity, freed and retracted lateralward and slightly upward with the fibers of the gluteus muscle. The periosteum and ligamentous attachments of the tuberosity are now boldly incised to the bone between the medial and the lateral hamstring muscles and the external rotator muscles of the hip. Subperiosteal stripping continues upward and downward as far as is necessary to remove all the involved bone. One can proceed to the acetabulum or to the pubis. The stripping is enhanced because of the thickening and edema of the periosteum usually present in osteomyelitic foci. No important structures are jeopardized if the dissection is continued strictly to the subperiosteal region, as these are all lifted away medially and laterally with the basic periosteal flaps. The bone can be removed piecemeal with the curet or rongeur or severed with a saw or mallet and chisel. In extensive lesions the entire bone may be lifted out of its bed. With ordinary care in sealing off the wound, rectal contamination does not occur. The patient may be constipated for several days after operation by the administration of opium.

REPORT OF CASES

Osteomyelitis of the Sacro-Iliac Joint.—Case 1.—L. B., a white boy aged 15 years, was admitted to the hospital on Dec. 16, 1931, with symptoms of six weeks' duration. The condition began suddenly, with headache, chills, fever and a dull

aching, nonradiating pain "back of the right hip," soon accompanied by diarrhea and urinary frequency. Objectively, there was fulness in the right lower abdominal quadrant. Moderate swelling and tenderness were noted over the right sacro-iliac region. Digital rectal examination revealed a tender fluctuating mass at the lower margin of the right sacro-iliac joint. The white cell count was 14,200. Roentgen examination confirmed the destructive nature of the sacro-iliac and the adjacent iliac lesion (fig. 1). A diagnosis of pyogenic osteomyelitis was made.

Operation.—Total resection of the sacro-iliac joint was done in January 1932. The periosteum of the ileum was found to be thickened, gelatinous and of a dirty grayish color. The bone beneath was injected and roughened. At the junction of the middle and the posterior third of the ilium, just below the crest on the pelvic surface, there was encountered a sequestrum measuring about 3 inches (7.6 cm.) in diameter. It lay loosely in a subperiosteal crater of pus and débris. Thick pus was present in the anterior part of the joint as well as another sequestrum. Most of the sacrum was necrotic, and considerable curettage was necessary to reach healthy bone. The immediate postoperative convalescence was uneventful.

Fig. 1 (case 1.)—Roentgenogram showing sacro-iliac osteomyelitis with sequestrums in the anterior margin of the joint and the posterior wing of the ilium. There is slight asymmetry of the pelvis.

The temperature was normal two weeks after operation. Staphylococcus aureus was recovered. The diagnosis in the pathologic report was pyogenic osteomyelitis. Complete healing occurred in about eighteen months.

Case 2.—R. C., a white man aged 21, was admitted to the hospital on Sept. 23, 1931, with symptoms of ten days' duration, which were of sudden onset, with a dull aching pain in the left groin, painful micturition and fever. The patient appeared acutely ill and septic. The left inguinal glands were enlarged and tender. A tender soft mass could be palpated in the lower part of the left side of the pelvis. There was suggestive tenderness over the left sacro-iliac region. A roentgenogram revealed blurring and increased condensation about the sacro-iliac joint. The white cell count was 16,500. General hygienic measures and traction were continued for several weeks until the diagnosis of pyogenic osteomyelitis was more certain and the operative risk less.

Operation.—Partial resection was done. The sacral portion was markedly necrotic. About 500 cc. of pus was evacuated from the pelvis. There was a moderate degree of shock. Staph. aureus was recovered, and inoculation of a

guinea-pig gave negative results. The diagnosis in the pathologic report was pyogenic osteomyelitis. The convalescence was uneventful, except for several metastatic abscesses in the soft tissue. Healing occurred in ten months (fig. 2 *B*).

CASE 3.—D. T., a white youth aged 17, was admitted to the hospital on Jan. 18, 1933, with symptoms of four months' duration, consisting of pain in the back and sciatica of acute onset. He had lost 28 pounds (12.7 Kg.) in weight. Two weeks before operation a spontaneous sinus appeared over the left buttock. There was bilateral spasm of the lumbar muscles, which, however, permitted considerable spinal motion. There was definite tenderness over the left sacro-iliac joint. The motions of the hip were free. The white cell count was 11,400. A roentgenogram showed a destructive lesion of the left sacro-iliac joint. A diagnosis of pyogenic osteomyelitis was made.

Operation.—Total resection was done. The chief destruction was found in the upper and anterior poles of the joint, involving the sacrum considerably. The

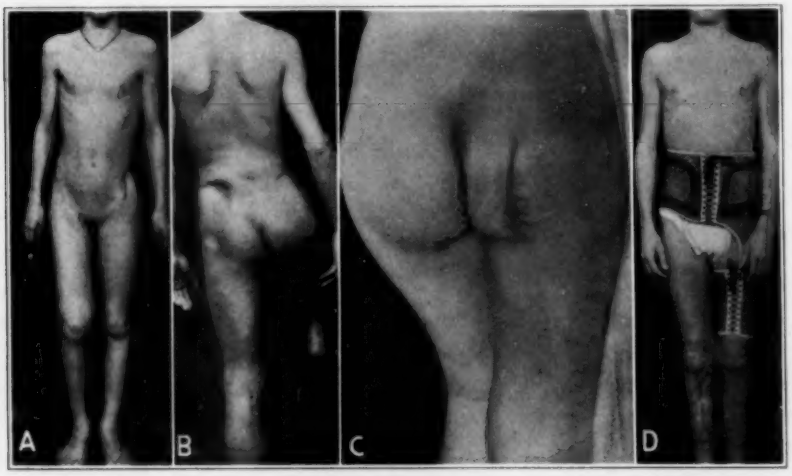

Fig. 2.—Photographs of patients in whom healing has occurred. *A* (case 10) shows a patient with healed osteomyelitis of the ilium; *B* (case 2), a patient with healed osteomyelitis of the sacro-iliac joint, demonstrating a negative reaction to the Trendelenburg test; *C* (case 15), a patient with healed osteomyelitis of the ischium, and *D*, a patient wearing the convalescent brace used for all pelvic foci.

ilium just above the sciatic notch was brittle. Only a few cubic centimeters of pus was encountered in the joint. The patient's recovery was uneventful. Four months later the sinus was again explored, and a small sequestrum was removed. Healing occurred in about one year. No bacterial organisms were recovered at operation. The diagnosis on the basis of the pathologic picture was chronic pyogenic osteomyelitis, with calcification of the joint surfaces.

CASE 4.—William S., white, aged 38, was admitted on Feb. 14, 1933, with symptoms of two and one-half years' duration. He had definite tenderness over the left sacro-iliac joint and a sinus just above the middle crest of the ilium. In January 1931 he was operated on by the Smith-Petersen method. The operative wound healed in four months after a stormy convalescence, but the original sinus never healed, nor was the patient relieved of his sciatica. A diagnosis of pyogenic osteomyelitis was made.

Operation.—Partial resection was done. The spines of the ilium were roughened and almost fused to the sacrum. The entire joint was destroyed, and the ilium was sclerotic and very thick. The sacral portion was necrotic at the level of the third segment, and the sacral canal was opened in eradicating this area. Blunt dissection of the soft parts of the pelvis led to an abscess. Several branches of the superior gluteal vein were accidentally torn but fortunately could be clamped and ligated. A moderate degree of shock followed. Staph. aureus and streptococci were recovered. The diagnosis on the basis of the pathologic picture was pyogenic osteomyelitis, with secondary productive changes in the joint. The postoperative convalescence was uneventful except for some intrapelvic hemorrhage. Several small transfusions of blood were given, followed by marked improvement in the general condition, and healing was sound in about six months.

Case 5.—Earnest B., aged 48, was admitted to the hospital in June 1933, with symptoms of four months' duration. The insidious onset was described as a "bellyache." An abscess soon appeared posteriorly. The patient lost some weight and could not bear weight because of pain in the right hip. Fourteen small sequestrums extruded spontaneously. There was a slight scoliosis to the left and a small sinus over the right sacro-iliac region. The skin about this was dull red and edematous. Infiltration and marked tenderness were elicited in this region. A roentgenogram showed an extensive destruction of the entire sacro-iliac joint. A diagnosis of pyogenic osteomyelitis was made.

Operation.—Total resection was done. The iliac flap was rapidly removed with the aid of a motor saw. There was brisk hemorrhage from the smaller vessels. The entire joint was necrotic, but no free pus was encountered. The diagnosis on the basis of the pathologic picture was pyogenic osteomyelitis, with no signs of specific inflammation. There was moderate shock, and the wound did not do so well as was expected. The drainage was profuse. The patient died at home about four months after operation, from an unknown cause.

Case 6.—Francis H., white, aged 20, had "silent" involvement of the left sacro-iliac joint of about six years' duration. He had multiple foci, including acute spondylitis, all of which necessitated drainage. The sacro-iliac joint was explored as a possible source of continued occasional metastatic foci and was considered to be the initial primary focus. A diagnosis of pyogenic osteomyelitis was made.

Operation.—Partial resection of the sacro-iliac joint was done in September 1933. The joint was found to be fused and obliterated for the most part. The posterior portion of the sacrum was friable. The ilium was dense and sclerotic. The pathologic process consisted of mild subacute nonspecific inflammatory changes. Healing occurred promptly.

Case 7.—Charles G., white, aged 17 years, had symptoms of one year's duration. There was chronic involvement of the lumbosacral and both sacro-iliac joints. The patient was a very poor operative risk as he was emaciated and covered with decubiti. Operation was performed as a last resort. A diagnosis of pyogenic osteomyelitis was made.

Operation.—Bilateral partial resection was done in February 1934. No frank pus was encountered, but considerable necrosis was present. Staphylococci were recovered. The patient died in about three months.

According to the autopsy report the diagnosis was subacute osteomyelitis of the lumbosacral portion of the spine, the sacro-iliac joints and the innominate bones and thrombosis of the right common iliac and left iliac veins.

Case 8.—Wayne M., white, aged 23, had metastatic involvement of the lumbosacral and both sacro-iliac joints of about eighteen months' duration. He was

very septic and ill. Retroperitoneal drainage was established through Petit's triangle, but he grew progressively worse. A diagnosis of pyogenic osteomyelitis was made.

Operation.—Partial resection of the sacro-iliac joint and evacuation of the retroperitoneal abscess were done in August 1932. The patient died one month later, after a stormy period.

According to the autopsy report the diagnosis was chronic suppurative arthritis of both sacro-iliac joints, osteomyelitis of the lumbosacral portion of the spine, abscess of the right psoas muscle, suppurative phlebitis of the left femoral vein, multiple metastatic abscess of the soft tissue, chronic osteomyelitis of the scapula, amyloidosis of the liver and spleen and septicemia.

Osteomyelitis of the Ilium.—Case 9.—Margaret F., white, aged 12 years, was admitted to the hospital in 1934 with symptoms of three years' duration. After a local injury she complained of intermittent pain and aching in the left hip and the region of the thigh. She had occasional night cries. She soon became acutely ill and feverish and had severe pain in the left hip and the region of the thigh. Incision and drainage of the thigh were soon followed by spontaneous drainage. The patient was a total invalid after the onset of the symptoms. Examination revealed a sinus in the upper anterior one third of the left thigh. Acute pain was elicited in this region on digital pressure. The hip was fixed in flexion and adduction of 75 degrees, associated with flexion contracture of the left knee of 75 degrees. The general condition was fair. The sedimentation rate was not rapid. The white cell count was 21,300, and the red cell count was 3,400,000. The roentgenogram showed destruction of the entire wing of the left ilium. A diagnosis of pyogenic osteomyelitis was made (fig. 3 *C* and *D*).

Operation.—Total resection was done in March 1934. The approach was difficult because of the contracture of the hip. The sinus led to the true pelvis just over the ascending ramus of the pubis, and pus was encountered when this depth was reached. Three major areas of destruction were found in the ilium (fig. 4). All the periosteal and soft structures were dense and adherent. After complete débridement a fairly dry field remained. Staphylococci were recovered, and the pathologic picture was that of pyogenic osteomyelitis. The most extensive change histologically was an irregular reactive osteosclerotic inflammatory process. The bone marrow consisted of hyperemic fibrous tissue which still showed some cellular infiltration. Destructive activity was only moderate, even in the region of the gross crater abscess, which was covered by scar, on the surface of which the granulations were necrotic. Numerous purulent foci were still present in the more superficial marrow spaces and in the neighborhood of the periosteal abscess, which contained necrotic spicules and was otherwise in an advenced stage of organization. A diagnosis of chronic osteomyelitis with extensive osteosclerosis surrounding periosteal abscess formation was made. The patient had a rather stormy convalescence, but healing occurred in about one year, with little or no regeneration of the iliac wing.

Case 10.—Gertrude G., white, aged 13 years, was admitted to the hospital in August 1933, with symptoms of two months' duration. The onset and course were subacute. The patient was fairly well nourished and walked with a limp on the left side. The left buttock was flat, and the left trochanter was less prominent than the right. A tender mass was palpated above the left hip joint, which exhibited limitation on rotation. The roentgenogram showed a destructive lesion of the iliac wing simulating tumor formation (fig. 3 *A* and *B*). A diagnosis of tumor or pyogenic osteomyelitis was made.

Operation.—Partial resection was done in August 1933. A tremendous amount of odorless edema and necrosis of the soft parts was encountered. The entire anterior two thirds of the upper half of the ilium was gone except for a small outer shell, which was sequestrated. Within the iliac fossa were found many spiculed sequestrums the size of tooth picks in a mass of débris. The pathologic picture was that of subacute and chronic fibrous osteomyelitis. Histologic examination showed a great amount of young cancellous bone with irregular texture. The bony trabeculae were composed of lamellar bone, fibrous bone and even calcified cartilage in the central portion. In part there was an osteoid layer with signs of inflammation. Between the bony trabeculae in several areas larger masses of hyaline cartilage were interposed, and there was continuous transformation of cartilaginous tissue toward fibrous bone tissue, as is commonly the rule in

Fig. 3.—Preoperative and postoperative roentgenograms of the pelvis in two cases of iliac osteomyelitis. *A* (case 10) shows an untreated lesion of over two months' duration, which suggests tumor formation rather than inflammation. *B* was made after partial resection. *C* (case 9) shows an untreated lesion of almost four years' duration. *D* was made after total resection. A subtrochanteric fracture was noted after manual correction of the flexion deformity of the hip, performed at the time of operation.

callous formation or in myositis ossificans. The surrounding soft tissues were represented by transverse striated musculature, which showed the highest degree of edematous infiltration. Among the exudate cells, lymphocytes and polymorphonuclear leukocytes were present. A diagnosis was made of nonsuppurative subacute osteomyelitis and productive periostitis with marked inflammatory edema of the surrounding musculature. There was no suggestion of tumor formation.

The patient fully recovered in four and one-half months.

Case 11.—Edna H., aged 11 years, was admitted to the hospital on May 13, 1935, with symptoms of ten days' duration. The onset was acute with pain in the right hip and groin associated with high fever, chills and vomiting. The child appeared to be acutely ill. The right hip was held in flexion. Considerable motion of the joint could be elicited, however, when her attention was distracted. The tenderness over the right groin was indefinite. In the right iliac fossa along the iliac crest backward definite tenderness and some muscle spasm were noted. The white cell count was 16,900, and the blood culture yielded staphylococci. In about five days a definite mass formed in the right iliac fossa, simulating an appendical abscess, and the temperature dropped to about 101 F. Serial roentgenograms revealed a supracotyloid destructive process (fig. 5). The diagnosis was pyogenic osteomyelitis as differentiated from acute appendicitis.

Operation.—Partial resection was done. A vertical incision from the iliac crest to the greater trochanter followed by blunt dissection exposed the supracotyloid region. The soft tissues were all edematous, and the outer cortex of the bone was soft and friable. No pus was encountered until the inner table was opened. Staphylococci were recovered, and the pathologic picture was that of acute pyogenic

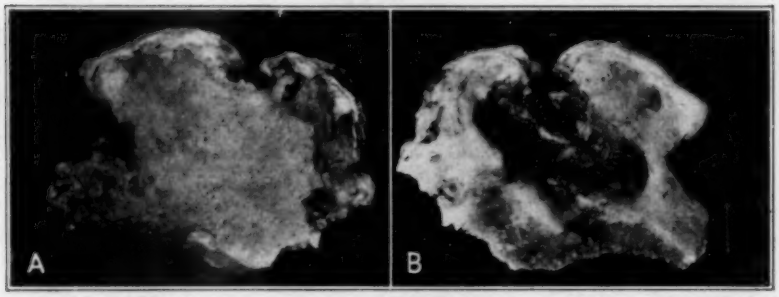

Fig. 4 (case 9).—Pathologic specimens of the iliac wing. *A*, the outer surface. The outer cortex was broken through in two places, one below the anterior superior spine and the second in the middle of the crest, and involved the cartilaginous apophysis as well as the bone. The bone was rather smooth and in many places was covered by densely adherent granulation tissue. The outer cortex formed a shell-like thin layer, whereas the inner cortex, so far as it was preserved, showed considerable thickening. *B*, the inner surface, showing a typical crater defect, with lateral displacement of the outer cortex.

osteomyelitis. The patient continued to have a low grade fever, which subsided only after counterincision and drainage along the iliac crest were performed. Healing occurred in about four months, with a residual ankylosis of the hip joint in good position.

Case 12.—John S., white, aged 41, was admitted to the hospital on Feb. 21, 1936, with symptoms of forty-one years' duration. The present flare-up followed a pathologic fracture of the right humerus. Local symptoms came on acutely with sharp pain about the right hip and flank, and a perirectal abscess presented soon afterward. This was incised and drained. The roentgenograms showed extensive honeycombing and destruction of the iliac wing, hip and trochanteric region (fig. 6). A diagnosis of pyogenic osteomyelitis was made.

Operation.—Partial resection was done on March 31. Numerous sequestrums were encountered. The main pathologic process in the ilium centered about the anterior third of the wing of the ilium. There was a typical crater-like destruction on the pelvic surface, with a considerable collection of débris and pus. A sinus had apparently burrowed downward into the true pelvis. The ilium was tremendously thickened. The hip joint itself was entirely denuded of cartilage and destroyed, but allowed manual correction. There was a moderate degree of postoperative shock. Four days later sepsis developed. The wound, which for one reason or another had been partially closed and inadequately drained, was opened and found filled with purulent exudate. The patient did not rally and died on April 12 of peritonitis, incident to a secondary infection with colon bacilli from the perianal fistula. This was the only case in which the Orr technic was not observed.

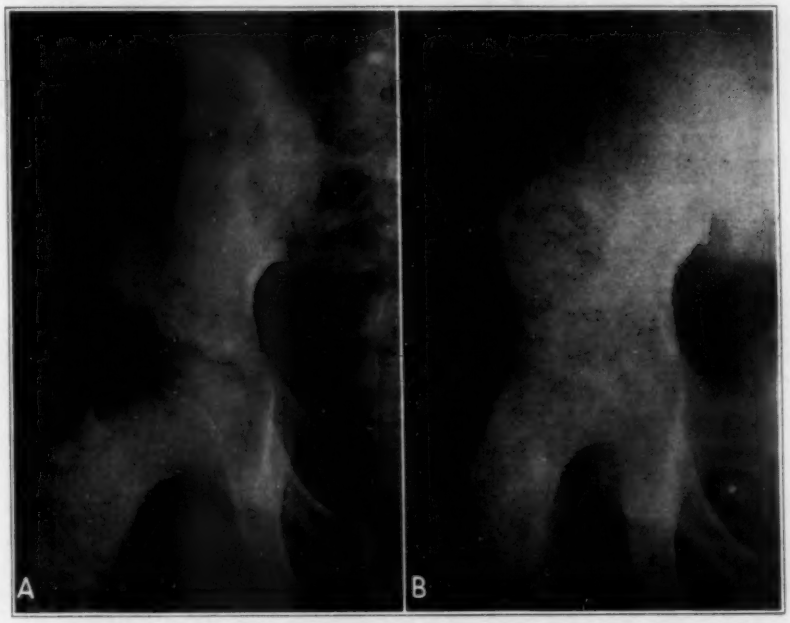

Fig. 5 (case 11).—*A,* roentgenogram of the pelvis, illustrating a prepuberty supracotyloid lesion twelve days after the onset of symptoms, which already involved the superior rim of the acetabulum. (The patient was referred by Dr. H. S. Conrad.) *B,* a roentgenogram taken two months later. The disease had extended to the wing of the ilium and some wandering of the acetabulum and head of the femur had occurred.

Case 13.—Gloria M., aged 11 years, was admitted to the hospital on April 24, 1936, with symptoms of three days' duration. She fell while roller skating and struck the left hip. That night she had a slight chill and fever, accompanied by anorexia and marked constipation. The fever and pain in the left hip persisted. On examination she was found to be mildly septic. The left hip was held in flexion and adduction, but was quite freely movable. There were marked tenderness over the lateral portion of the ilium and pubis and some tenderness in the left iliac

fossa. The patient was placed in traction, and general measures were instituted. The white cell count was 6,000, and the sedimentation rate was rapid. The fever was sustained. On the fifth day definite infiltration appeared below the anterior crest of the ilium. The roentgenogram was still normal (fig. 7 *A*). The general condition was improved. A diagnosis of pyogenic osteomyelitis was made.

Operation—On April 29 an external subperiosteal abscess was drained just below the anterior crest of the ilium. The bone was very hemorrhagic but appeared slightly pale at the anterior metaphysial border. A fragment was removed for examination. The pathologic report was acute osteomyelitis, and staphylococci were recovered. A subacute course followed, and in about two weeks roentgenographic delineation of the lesion was noted (fig. 7 *B*), and partial resection (May 11) of the ilium was performed. The supracotyloid region was

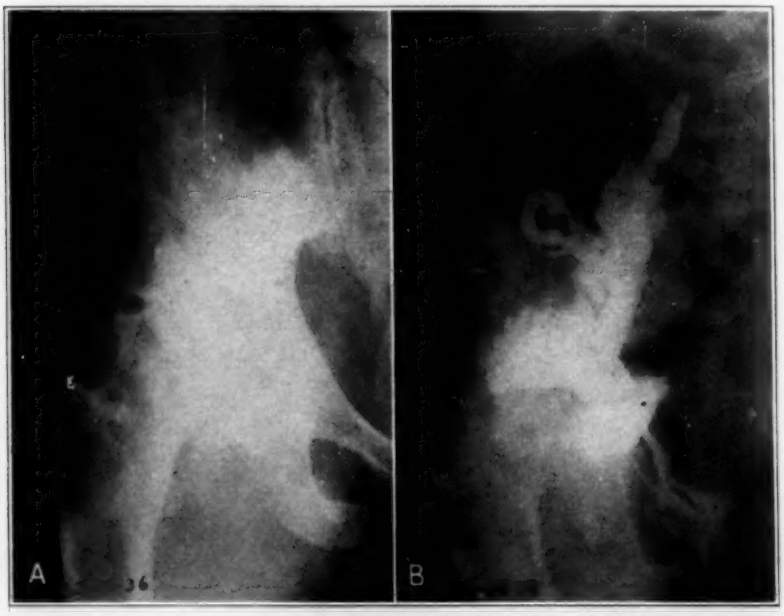

Fig. 6 (case 12).—Roentgenograms showing an iliac lesion involving the hip joint and femur of forty-one years' duration with a recent flare-up. The injection of iodized poppy-seed oil 40 per cent into the perirectal sinus revealed the extensiveness of the soft tissue abscess and the dangers of secondary infection from the rectum, especially after radical operation. (The patient was referred by Dr. Paul Forgrave.)

not involved, and an iliac abscess was now encountered and drained. Complete recovery followed subsequent treatment of a "silent" tibial metastatic lesion. Healing occurred in about five months.

Osteomyelitis of the Ischium.—Case 14.—Russel C., aged 16 years, was admitted to the hospital on Nov. 2, 1933, with symptoms of eight weeks' duration. The onset began suddenly, with pain in the left hip, limpness and fever. This was aggravated by sitting and jarring. The patient had a series of boils three weeks previously. He had lost 27 pounds (12.2 Kg.) in weight. The leukocyte count

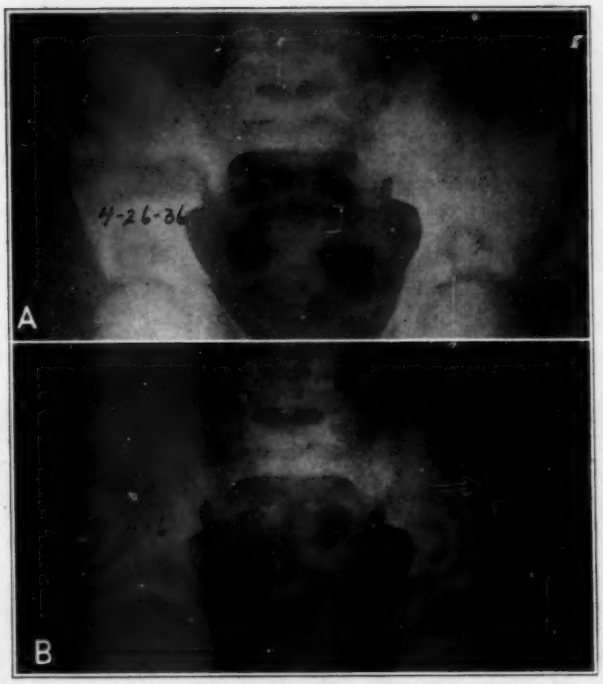

Fig. 7 (case 13).—*A*, roentgenogram of the pelvis five days after the onset of symptoms. No abnormal changes are demonstrated. *B*, a roentgenogram made twenty-one days after the onset. Destructive and productive changes appear at the lateral margins of the left ilium.

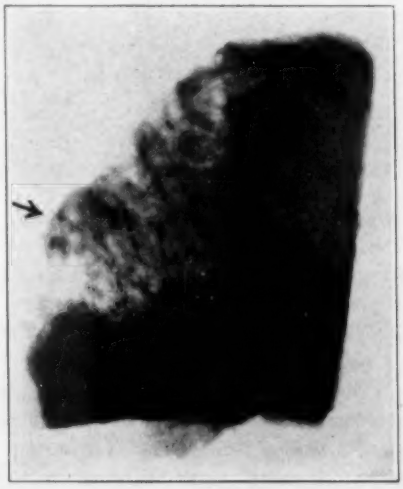

Fig. 8 (case 13).—Roentgenograms of the resected portion of the ilium twenty-two days after the onset of symptoms. The destructive process about the secondary epiphysial centers of activity is evident, although the child was only 11 years of age. The arrow points to the anterior superior spine. (Presented by courtesy of Dr. A. B. McGlothlan.)

was 14,000. There were occasional radiating pain in the left knee, and the hip and knee had drawn up. Examination revealed a well developed but moderately emaciated boy. The left lower extremity was held in 90 degrees of external rotation and 50 degrees of flexion. He held the left hip guardedly. The inguinal glands were enlarged, and there were no local signs of inflammation but some induration about the left ischiorectal fossa and definite tenderness over the left ischial tuberosity. Rectal examination revealed tenderness of the left ischium. The white cell count was 10,000. The roentgenograms showed a marked honeycombing and destructive process in the left ischium (fig. 9 *C* and *D*). A diagnosis of tumor or pyogenic osteomyelitis was made.

Operation.—Total resection was done in November 1933. There was no frank pus, but considerable necrosis and marked parosteal edema were present. There

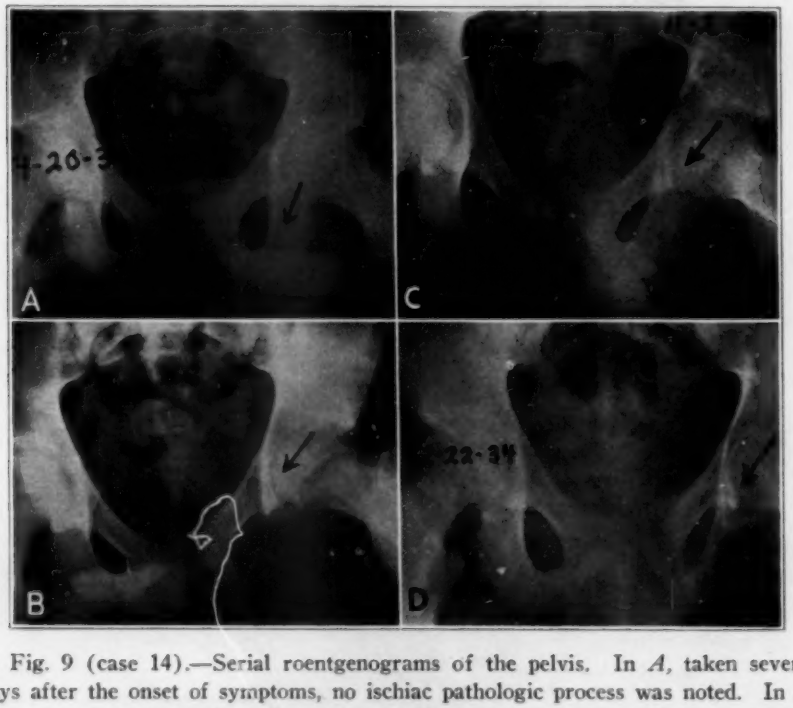

Fig. 9 (case 14).—Serial roentgenograms of the pelvis. In *A*, taken several days after the onset of symptoms, no ischiac pathologic process was noted. In *B*, taken about eight weeks after the onset of symptoms, the pathologic changes in the ischium simulated tumor rather than an inflammatory lesion. *C* was taken immediately after resection of the ischium and *D* about four months later, when regeneration was taking place.

was no periosteal proliferation. The bone was hemorrhagic and shaggy and strongly suggested tumor formation. Staphylococcus aureus was recovered from the necrotic bone, and osteomyelitis was diagnosed pathologically. Histologic examination showed necrotic marrow and some cortical sequestration, but the cartilaginous portion of the ischium was well preserved. In spite of the predominance of bone absorption, production of new bone was nevertheless occurring. In some places the marrow was densely infiltrated by polymorphonuclear leuko-

cytes. The granulations rapidly changed into dense fibrous tissue, with almost immediate hyaline degeneration. While no frank pus was visible, there was nothing to indicate tumor formation.

The convalescence was uneventful. The wound healed in four months. The external rotation deformity of the hip present on admission awaits corrective osteotomy.

CASE 15.—Hazel M., white, aged 6 years, was admitted to the hospital on Feb. 4, 1934, with symptoms of two days' duration. She began to limp suddenly two days before her admission to the hospital. She became ill rapidly and had a high fever, the temperature reaching 106 F. Several days before the onset she had an infected blister on the foot. On admission she was flushed and almost moribund. The hip was held in flexion but could be moved substantially. Pressure over the ischium on rectal examination caused the patient to wince even though she was practically unconscious. Aspiration in the region of the ischium with the patient under anesthesia gave negative results. (A tentative diagnosis of pyogenic osteomyelitis was made at this time.) The course was stormy, and several pyemic abscesses and also metastasis of the right fibula developed. In about six weeks the ischiatic lesion was definitely noted in serial roentgenograms (fig. 10), and the vulva was swollen.

Operation.—Total resection and removal of the floor of the acetabulum were done. On exploration medially and up toward the pubis, several cubic centimeters of frank pus were exuding from this region. The entire ischium was roughened. This reaction impressed one as being of a collateral nature. That portion of the ischium uniting with the descending portion of the pubis lifted out easily but did not appear to be definitely sequestrated. The floor of the acetabulum was, however, necrotic. Moderate shock ensued. Staphylococci were recovered, and the pathologic picture was that of pyogenic osteomyelitis. The changes in the infra-acetabular portions of the bone revealed changes chiefly of a collateral ischemic nature. The fibrous bone showed active bone formation leading to osteosclerosis. The cortical bone was alive, whereas the spongy bone showed necrosis. The acetabular region, the initial and primary seat of involvement, showed entire areas where the old spongy bone had disappeared and were occupied by dense fibrous bone, in greatest part osteoid. The small remnants of fatty bone marrow still showed inflammatory cellular infiltration. A diagnosis of subacute and chronic nonsuppurative osteomyelitis was made (osteomyelitis following invagination of old necrotic spongy bone by new bone apposition). The convalescence was uneventful, and the wound healed in three months with regeneration of the ischium (fig. 2 *C*).

CASE 16.—Wanda S., aged 13 years, had symptoms of three months' duration, which were of subacute onset and course. On admission of the patient to the hospital it was difficult to localize the trouble accurately. In March 1933 the left hip was drained through a lateral incision, and no definite pathologic process was noted. In August 1933 the upper anterior portion of the ilium was exposed, and only sclerotic bone was encountered. The clinical course continued to show the presence of an active focus, which was finally located in the left ischium, after the vulva became edematous.

Operation.—Total resection was done in November 1933. Through the usual approach the entire ischium was found to be necrotic and in a bed of pus. Staphyloccoci were recovered, and the pathologic diagnosis was chronic productive periostitis, subacute osteomyelitis and arthritis. The convalescence was uneventful, and the wound healed in three months, at which time the ischium had regenerated

CASE 17.—Jule L., aged 16 years, was admitted to the hospital on July 22, 1935, with symptoms of eighteen days' duration. The onset was subacute and followed an indirect injury to the left hip. There was fever but no chills, and the pain was referred to the left hip and occasionally to the left knee medially. The patient appeared to be acutely ill. The left hip was held in flexion and some external rotation. Gentle manipulation elicited a fair range of motion, however. The entire left buttock was tense and tender and warm. Rectal examination

Fig. 10 (case 15).—Serial roentgenograms of the pelvis in a child, beginning three days after the onset of symptoms. Slight infra-acetabular changes were noted fourteen days later (2/19/34), but convincing evidence of destruction did not appear until thirty-six days after the onset of symptoms (3/10/34). The next picture was made after resection of the ischium and the last picture reveals regeneration about four months after operation.

revealed maximum tenderness over the ischium. The white cell count was 27,000, and the blood culture was negative. The roentgenogram is shown in figure 11. A diagnosis of pyogenic osteomyelitis was made.

Fig. 11 (case 17).—Roentgenogram of the pelvis taken eighteen days after the onset of symptoms. The shadow of the soft tissue abscess predominates and obscures the skeletal ischiac changes. (The patient was referred by Dr. H. S. Forgave.)

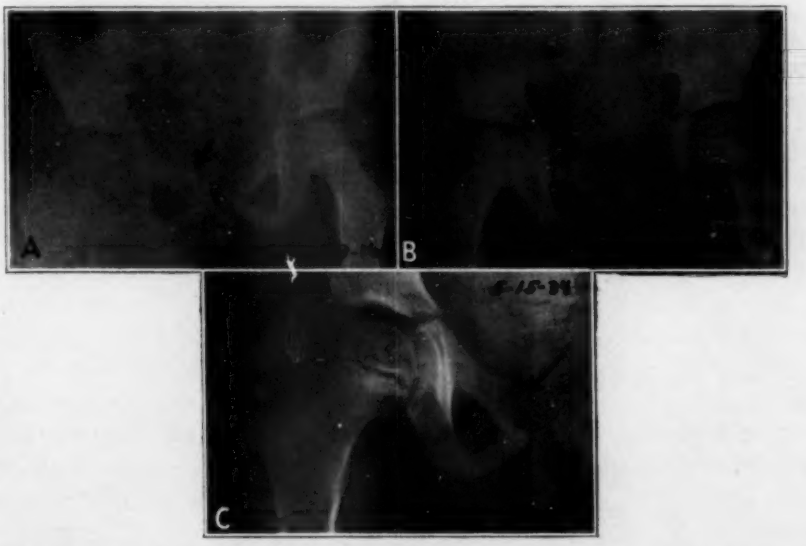

Fig. 12. (case 18).—*A*, roentgenogram showing a juxta-epiphysial pubic lesion three weeks after the onset of symptoms. The abscess and flexion of the hip blur the skeletal outlines. *B*, roentgenogram taken three weeks after operation. *C*, roentgenogram taken about six weeks later. There had been no extension, and the curetted bone is clear in outline.

Operation.—Total resection was done. A huge subgluteal collection of pus which involved the subgluteal bursa was evacuated. The ischium was bare owing to the stripping action of the pus. Most of the destruction was at the tuberosity, but the entire bone was removed because of the extensive purulent dissection already present. Staphylococci and streptococci were recovered. The pathologic diagnosis was subacute and acute pyogenic osteomyelitis. In spite of the radical resection, the patient's course continued to be of the low grade septic type, until the floor of the acetabulum was removed for more adequate drainage. The convalescence was then good, and healing occurred in about six months. There was residual ankylosis of the hip in good position.

Osteomyelitis of the Pubis.—Case 18.—Phyllis S., aged 5 years, was admitted to the hospital on March 16, 1934, with symptoms of three weeks' duration. The condition was of sudden onset, with severe pain in the right groin associated with fever. She remained in bed with the right hip drawn up. She lost from 10 to 15 pounds (4.5 to 6.8 Kg.) in three weeks and appeared to be acutely ill and septic. The temperature was 104 F. There were marked tenderness over the inguinal ligament and some swelling of the right mons pubis and also the vulva of a lesser degree. The right hip was acutely flexed, but otherwise was normal. When the patient's attention was diverted, the hip could be moved. The white cell count was 32,000. The rectal examination (under anesthesia) revealed definite infiltration in the pubic region, the descending ramus and the medial wall of the right side of the pelvis. When the thigh was extended a fluctuating mass appeared in the right groin. The diagnosis made was pyogenic osteomyelitis as differentiated from appendical abscess.

Operation.—Drainage and curettage were done in March 1934. Through a curved incision about 3½ inches (8.9 cm.) long, cutting obliquely across the medial half of Poupart's ligament, an abscess cavity was entered just above Poupart's ligament and about 50 cc. of an odorless brownish creamy pus was found. Digital exploration showed this to be a granulation-lined subcutaneous abscess. About 100 cc. of thick green pus kept welling up from below Poupart's ligament, and this was found to be coming from Scarpa's triangle. Further digital examination of the right pubis showed a small localized necrotic area at the chondro-osseous border portion of the body of the pubis. This was carefully curetted out. Staph. aureus was recovered. The pathologic picture was that of acute osteomyelitis. Healing occurred in six weeks after an uneventful convalescence.

CIRCULATORY DISTURBANCES REFLEXLY INAUGURATED BY STIMULATION OF THE CELIAC PLEXUS

A PRELIMINARY REPORT

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Circulatory disturbances coincident with surgical manipulations in the upper part of the abdomen have frequently been described. A transitory initial rise in the blood pressure succeeded by a fall as a rule follows manipulation of the abdominal viscera. Recently, in a case in which a difficult cholecystectomy was performed, at the stage when operative manipulation was maximum, an unusual phenomenon was observed in the course of routine sphygmomanometry by the auscultation method. The blood pressure, which had remained constantly within normal limits with a difference of 60 mm. of mercury between the systolic and the diastolic level, suddenly became unobtainable for twenty minutes, although the peripheral pulse could still be palpated and was not materially altered in rate. At first, the mercury column being allowed to fall very slowly, a single beat was heard at about the level of the previous systolic pressure. Within a few minutes this single beat could not be obtained. It was further noted that when the operative manipulations had ceased, the normal auscultation sounds were again obtained and at a level comparable to the previous determinations. Thereafter, careful and frequent determinations on the blood pressure were made during all operations on the upper part of the abdomen. The phenomenon described was soon obtained in the course of another cholecystectomy and during gastric resection. The patients had been given morphine and atropine preoperatively. In an attempt to account for the circulatory disturbance noted, observations were made in the laboratory to determine the effects on the blood pressure curve obtained during intra-abdominal manipulations.

EXPERIMENTAL STUDY

Dogs were used for the experiments, and attempts were made to reproduce the same operative conditions. Preoperative medication consisted of 5 mg. of morphine sulfate and 0.1 mg. of atropine sulfate per kilogram of body weight

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administered subcutaneously one hour before anesthesia. Ether anesthesia was then induced by the open cone method, and an endotracheal tube was passed through the mouth into the trachea and made air tight by inflating a cuff surrounding it. This tube was connected to a rebreathing bag, and a canister containing soda lime was inserted between the two so that the technic of carbon dioxide absorption [1] could be maintained. Ether and oxygen were supplied to the system by means of a Foregger metric machine. The blood pressure was recorded from a cannula inserted into the femoral artery and connected to a mercury manometer which registered on a kymograph. Respirations were recorded at the same time by means of a pneumograph applied about the thoracic region.

The peritoneum was opened by a long midline incision. Traction on the various viscera and intra-abdominal exploration resulted in circulatory disturbances consisting of a small rise in the mean arterial blood pressure and a slight reduction in the pulse pressure. Traction on the gallbladder, as shown by Brewer and Luckhardt,[2] was followed by apnea (chart 1 *B*), and a slight rise in the blood pressure was recorded. Exploration in the region of the solar plexus and

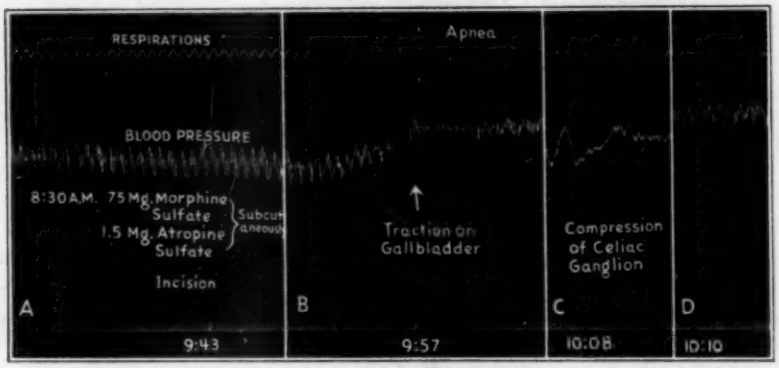

Chart 1.—Serial sections from an experiment on dog 34, which weighed 15 Kg. At 8:30 a. m. 75 mg. of morphine sulfate and 1.5 mg. of atropine sulfate were administered subcutaneously. At 9:43 a. m., when the initial incision was made, the blood pressure was normal, as indicated in *A*. In *B* are shown the effects of traction on the gallbladder at 9:57 a. m., which included apnea, a rise in the mean blood pressure and a reduction in the pulse pressure. Compression of the celiac ganglion at 10:08 a. m. brought about a marked reduction in the pulse pressure and no appreciable change in the mean blood pressure, as shown in *C*. After the compression of the celiac ganglion was released at 10:10 a. m., the pulse pressure recovered, as shown in *D*.

especially compression of the celiac ganglion gave an immediate response characterized by a marked reduction in the pulse pressure without a fall in the mean blood pressure (chart 1 *C*). The diminution of the range between the systolic

1. Waters, R. M.: Utility of Carbon Dioxide Absorption from Anesthetic Mixtures, Anesth. & Analg. **3**:20 (Feb.) 1924.

2. Brewer, N.; Luckhardt, A. B.; Lees, W. M., and Bryant, D. S.: Reflex Closure of the Glottis by Stimulation of Afferent (Visceral) Nerves, Anesth. & Analg. **13**:257, 1934.

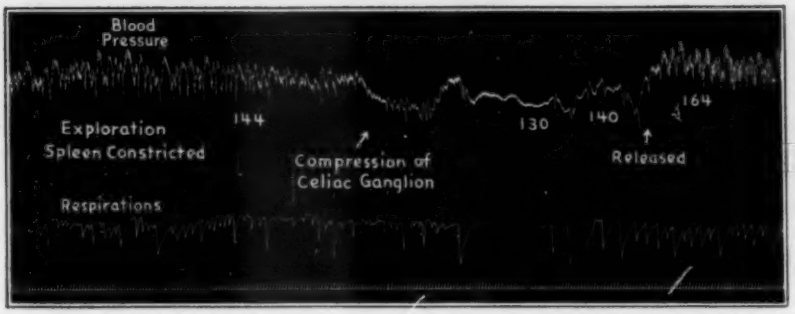

Chart 2.—A tracing showing the reflex in its entirety. Atropine sulfate was given previously.

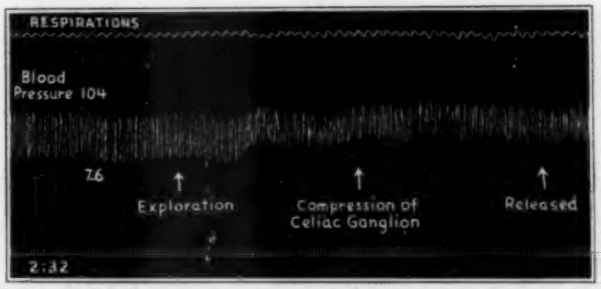

Chart 3.—A tracing showing the reflex when atropine sulfate was not administered.

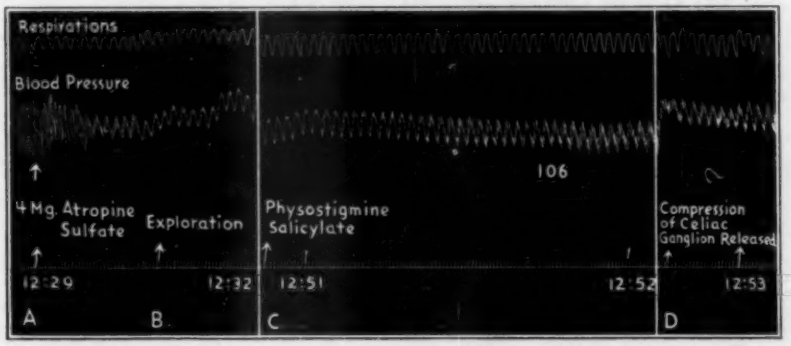

Chart 4.—Serial sections from an experiment on dog 201, which weighed 22 Kg. One hundred milligrams of morphine sulfate was administered at 10:30 a. m. In *A* is shown the immediate reduction of the pulse pressure after the intravenous injection of 4 mg. of atropine sulfate at 12:29 p. m. Intra-abdominal exploration at 12:32 p. m., indicated in *B*, was followed by compression of the celiac ganglion. The intra-abdominal exploration caused a reduction in the pulse pressure and a rise in the mean blood pressure, while compression of the celiac ganglion caused further and more marked reduction in the pulse pressure and no change in the mean blood pressure. After twenty minutes with no improvement in the pulse pressure, an intravenous injection of physostigmine salicylate at 12:51 p. m. readily caused a return of the pulse pressure, as shown in *C*. Intra-abdominal exploration was followed by compression of the celiac ganglion after the administration of physostigmine salicylate at 12:53 p. m., as indicated in *D*. The first procedure caused only a slight reduction in the pulse pressure and a slight rise in the mean blood pressure. The latter procedure caused a very slight further reduction in the pulse pressure and no change in the mean blood pressure.

and the diastolic pressure became so marked that the pulse pressure practically disappeared. At the same time the abdominal muscles became more tense, and the intestines protruded from the wound. On releasing the compression the pulse pressure returned (chart 1 *D*). Similar tracings were secured following the application and release of pressure on the celiac ganglion (chart 2). This reaction was repeated in several dogs prepared and experimented on in the same manner.

It was further noted that when atropine sulfate was not administered the production of this reflex was either absent or greatly diminished, the pulse pressure falling but slightly (chart 3). However, if a dog which had not received atropine sulfate and did not show this reflex was given an intravenous injection of the drug a definite reduction in the pulse pressure (chart 4 *A*) would immediately result, and compression of the celiac ganglion would then be followed by a further reduction in the pulse pressure (chart 4 *B*), which would be maintained for a long time. Furthermore, if physostigmine salicylate was given intravenously the pulse pressure was markedly increased (chart 4 *C*). After physostigmine salicylate was administered, efforts to elicit this celiac plexus reflex would result in only a slight reduction of the pulse pressure (chart 4 *D*).

It was therefore evident that compression of the celiac ganglion caused a reflex reduction in the pulse pressure and that paralyzing the vagus nerves with atropine sulfate intensified the reflex, whereas stimulation of these nerves with physostigmine salicylate prevented entirely or reduced an already existing reflex. It is interesting to note that Capps and Lewis,[3] in their work on pleural reflexes, have found that stimulation of the pulmonary vagus nerves (instead of their paralysis) is followed by a "cardio-inhibitory reflex, in which the heart is slowed and the pulse tracings make violent excursions, with a great range between systolic and diastolic pressure."

Further experimental studies to show the mechanism of this reflex are in progress and will be reported at a later date.

SUMMARY

Stimulation of the celiac plexus when atropine sulfate has been previously administered produces a marked reduction in the pulse pressure with maintenance of the mean arterial blood pressure.

Stimulation of the celiac plexus when atropine has not been administered previously causes little or no change in the pulse pressure.

Manipulations in the upper abdominal cavity cause a reduction in the pulse pressure, especially when atropine sulfate has been given previously.

Administration of physostigmine salicylate reduces the reflex.

477 First Avenue.

3. Capps, J. A., and Lewis, D. D.: Pleural Reflexes, Surg., Gynec. & Obst. **7**:243, 1908.

SIXTY-THIRD REPORT OF PROGRESS IN ORTHOPEDIC SURGERY

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CONGENITAL DEFORMITIES

Congenital Dislocation of the Hip.—Van Gorder [1] reviews the end-results in 58 cases of dislocation of the hip (73 hips) in which treatment was given at the Massachusetts General Hospital during the period from 1923 to 1933. Sixteen of the hips were treated by closed reduction. The results in 10, or 62 per cent, were rated as excellent; i. e., there were a negative Trendelenburg sign, normal motion and no limp, shortening or atrophy. The results in 4, or 25 per cent, were rated as good; i. e., there were a slight limp, a positive Trendelenburg sign, excellent function and no disability. The results in 2, or 12.5 per cent, were rated as fair. Fifteen hips were treated by open reduction. The results in 1 were rated as excellent and in 3 as poor. Bony ankylosis occurred in 2, and a stripping of all structures from the neck and trochanters was done on the last 2 hips preliminary to heavy traction and open reduction. The 8 hips treated by open reductions plus a shelf operation showed no excellent results; the results in 2 were rated as good, in 1 as "fair" and in 2 as poor, and bony ankylosis occurred in 3. Sixteen hips treated by

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This report of progress is obtained from a review of 144 papers selected from 296 titles relating to orthopedic surgery and appearing in the medical literature approximately between Nov. 1, 1936, and March 1, 1937. Only those which indicated progress were selected for review.

1. Van Gorder, G. W.: New England J. Med. **215**:613 (Oct. 1) 1936.

a shelf operation alone showed no excellent results; the results in 8, or 50 per cent, were rated as good, in 4, or 25 per cent, as fair and in 4 as poor.

Summarizing these statistics in a different way: Good functional results were obtained in 88 per cent of the hips treated by closed reduction, in 27 per cent of those treated by open reduction, in 25 per cent of those treated by open reduction plus a shelf operation and in 50 per cent of those treated by a shelf operation alone. While the author feels that these results are far from satisfactory, he points out that two thirds of the patients in this study were over 5 years of age when treated.

Congenital Hallux Valgus.—Hoffmann [2] reports 4 cases of congenital deviation of the second phalanx of the great toe and was able to find 4 similar cases described in the literature. The author considers the condition as a congenital arrest in development in the epiphysis of the phalanx. Correction was obtained by a wedge osteotomy of the proximal phalanx of the big toe.

Brittle Bones and Blue Scleras in 5 Generations.—Blue scleras, fragile bones, a tendency to deafness and marked relaxation of the ligaments constitute a clinical entity according to Hills and McLanahan.[3] Data concerning 51 persons in 5 generations, all traced back to a common female ancestor, revealed that 27, or nearly half, were affected; 60 per cent were females. Deafness was manifest in 57 per cent. The blue sclera is generally attributed to thinness of the sclerotic coat, through which the choroid shines. The fractures are not likely to be present at birth but appear throughout childhood until puberty is reached. Union is prompt and complete. The etiology is unknown, but the fact that the fractures appear to cease at puberty would lead one to suppose that approach along endocrine lines offered greatest promise.

DISTURBANCE OF GROWTH

Tibia Vara.—Thirteen new cases and reports of 15 collected from the literature are reviewed by Blount [4] to illustrate the occurrence of an osteochondritis similar to coxa plana and Madelung's deformity but located at the medial side of the proximal tibial epiphysis. The resulting abrupt angulation into varus with back knee and internal rotation of the leg is usually confused with rickets. The roentgenographic picture and the pathologic changes are like those of coxa plana and similar to those of chondrodysplasia but are quite different from those of rickets. The changes may appear in the first year or two of life (infantile type) as a

2. Hoffmann, H.: Ztschr. f. Orthop. **65**:353, 1936.

3. Hills, R. G., and McLanahan, S.: Brittle Bones and Blue Scleras in Five Generations, Arch. Int. Med. **59**:41 (Jan.) 1937.

4. Blount, W. P.: J. Bone & Joint Surg. **19**:1, 1937.

developmental exaggeration of the normal, with sloping epiphysis and beaklike, recurving metaphysis. Similar deformity may occur just before puberty (adolescent type), secondary probably to local trauma or possibly to infection. The age at which the deformity is observed is more important than the causative factor in determining the roentgenographic appearance. The roentgenographic picture presented by the infantile type gradually changes to that presented by the adolescent type, so that later the two can be distinguished only by the history. Treatment should be directed toward the mechanical relief of strain until the deformity is stationary or until the epiphysis is closed. Simple osteotomy is desirable in the correction of marked deformity. If it is done before the angulation becomes stationary, some degree of recurrence may be anticipated.

TUBERCULOSIS

Multiple Arthritis in Tuberculous Subjects.—Collins and Cameron [5] sound a warning against the too early acceptance of a tuberculous etiology in cases of insidious arthritis occurring in presumably tuberculous patients, as this often results in prolonged and expensive immobilization, which is prejudical to subsequent recovery of function. They suggest that in all such cases expectant treatment should be adopted. This should consist of maintenance of general hygiene and rest in bed without immobilization until unequivocal evidence of the tuberculous or nontuberculous nature of the joint appears or is obtained by examination or by biopsy of material obtained by aspiration.

Glutathionemia in Tuberculosis of Bones.—Altschuler [6] studied the glutathione content of the blood from children with tuberculosis of the bones and joints. The more severe the tuberculous lesion, the higher were the values for glutathione in the blood. The author feels that fluctuations in the glutathione content of the blood assisted in estimating the course of the disease. A review of the literature is given.

Result of Spinal Fusion in Tuberculous Spondylitis.—Grigorescu and Vasiliu [7] report on 82 of 182 cases of tuberculous spondylitis in which an Albee fusion operation was performed. Postoperative fixation consisted of three months of rest in a plaster bed followed by the use of back supports. After six months the patients were permitted to return to their occupation. Twenty-four were well from seven to nine years after the operation; healing was taking place in 50 with considerable improvement from three to four years after the operation. In eight of those with abscesses, the abscesses had healed. In 2 there was no change and the abscesses persisted. One with paraplegia was somewhat

5. Collins, D. H., and Cameron, C.: Brit. J. Surg. **24**:272, 1936.
6. Altschuler, M. M.: Acta tuberc. Scandinav. **10**:370, 1936.
7. Grigorescu, I. I., and Vasiliu, A.: Beitr. z. klin. Chir. **164**:1, 1936.

improved. Four had died from three months to five years after the operation. One died of sepsis four days after the operation.

Use of Biopsy Specimens Obtained by a Punch in the Diagnosis of Tuberculosis of the Knee Joint.—In connection with the arthroscope, which resembles a cystoscope and is used for direct observation of the knee joint, Burman and his co-workers [8] have developed a punch for obtaining biopsy specimens from the knee joint. It is inserted through the same puncture hole used for the arthroscope. The specimen is secured blindly, although the arthroscope is first used for localization. In 8 cases of proved tuberculosis of the knee, the diagnosis was made in 6 on the basis of specimens secured with this instrument. This method is offered as another diagnostic test for tuberculosis of the knee and is considered by the authors as preferable to the radical method of securing a biopsy specimen, as advocated by Hibbs.

OSTEOMYELITIS

Osteomyelitis of the Bones of the Hand.—Osteomyelitis of the bones of the hand secondary to infection of the soft tissue is often treated radically by guttering or by removal of the bone. Koch,[9] whose experience is extensive, condemns such radical surgical methods. He states:

> The essential factors in the treatment of osteomyelitis of the bones of the hand are adequate drainage of the overlying soft parts, cleanly surgical care, and avoidance of trauma—by irritating chemicals, by addition of infection from without, and, particularly, by the use of curette and chisel. If death of bone takes place a line of demarcation forms and the necrotic bone, if it is not extruded spontaneously, can be removed without trauma. To attempt to determine a line of demarcation by surgical intervention too frequently results in destruction of living bone and extension of infection, and often makes recovery of the affected bone impossible.

POLIOMYELITIS

Humoral Antibodies and Resistance to Poliomyelitis.—Sabin and Olitsky [10] found that monkeys recovering from a paralytic attack of poliomyelitis acquired humoral antibodies slowly two months after the attack. Convalescent monkeys showed resistance to reinfection by the nasal route long before antibodies appeared in their serum. Vaccinated monkeys often remained susceptible to nasal infection when their serum neutralized the virus. Humoral antibodies do not seem to have the same significance in vaccinated monkeys as in monkeys recovering from an attack of poliomyelitis.

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8. Burman, M.; Finkelstein, H., and Mayer, L.: Am. Rev. Tuberc. **34**:663, 1936.

9. Koch, S. L.: Surg., Gynec. & Obst. **64**:1, 1937.

10. Sabin, A. B., and Olitsky, P. K.: J. Exper. Med. **64**:739, 1936.

CHRONIC ARTHRITIS

Fibrositis.—Slocumb [11] states that "fibrositis" is the commonest form of acute or chronic "rheumatism." The term appears in the foreign literature frequently but is seldom seen in the American literature. The author, nevertheless, believes that a condition exists which should be referred to as "fibrositis" rather than by any other term. Although in most cases the exact etiology is unknown, fibrositis is believed to be due to some unidentified infection or toxin. He believes that primary fibrositis should include (1) "myositis;" (2) "bursitis;" (3) periarticular fibrositis, or so-called "capsular rheumatism;" (4) tendinous fibrositis, which leads to Dupuytren's contracture of the palm; (5) perineural fibrositis, of which "sciatica" and "brachial neuralgia" are the common types, and (6) panniculitis. In the first stage of the process there is a low grade inflammatory serofibrinous exudate with proliferating fibroblasts and newly formed blood vessels; in the second stage, indurated, tender thickened tissue or subcutaneous nodules may be produced which may show dense inflammatory hyperplasia of fibrous tissue, thickened blood vessels and inflamed nerve fibrils without leukocytic reaction; in the third stage, when the inflammatory stage has passed, indurations may disappear or remain as painless nodules or capsular thickening which may affect such regions as the shoulders or the palms. The author feels that the differential diagnosis between these two conditions lies in the abundant indirect evidence of the persistent integrity of the joint cavity in fibrositis; that is, the disease is at the joint and not within the joint. Laboratory findings revealed little or no change in the sedimentation rate in cases of periarticular fibrositis in contrast to the rapid rate in cases of atrophic arthritis. Likewise, roentgenographic findings are demonstrated only in cases of atrophic arthritis. Finally, the author feels that when objective changes, systemic manifestations and alterations of laboratory findings, known to be relatively consistent in cases of atrophic arthritis, remain persistently absent, the arthralgia, stiffness and articular thickening can be, with no little confidence, ascribed to periarticular fibrositis, and the fear of impending arthritis can be laid aside.

THE SHOULDER

Scaglietti [12] discusses obstetric lesions of the shoulder thoroughly and describes the method of therapy as used by Putti. The article is based on a study of 199 cases in which therapy was given at the Instituto Rizzoli from 1899 to July 1935. The condition was unilateral in 93 per cent of the cases and bilateral in 6.5 per cent. In 62 per cent of the total number the right shoulder was involved. Conservative treat-

11. Slocumb, C. H.: J. Lab. & Clin. Med. **22**:56, 1936.
12. Scaglietti, O.: Chir. d. org. di movimento **22**:183, 1936.

ment was used in 75 per cent of the cases and surgical treatment in 25 per cent. The cases were divided into three groups: those of obstetric trauma of the joint or the superior humeral epiphysis or distortion of the joint or fracture of the clavicle (62 cases), those of pure obstetric paralysis consisting of trauma to the nerve root, subdivided into paralysis of the upper part of the arm, the lower part of the arm and the whole arm (22 cases) and a mixed group of cases in which both articular damage and injury to the nerve were present (14 cases). In 101 cases of the series there was no record as to the exact type. The classification of these injuries depended on the results of the physical, roentgenographic and electrical examinations of the muscles. The roentgenographic studies showed that in the group of cases of trauma to the joint the normal angle of the head of the humerus as superimposed over the condyles was increased from a position of internal rotation of 20 degrees to an abnormal one of 40 degrees or more. Therefore, with the head of the humerus pointing posteriorly and the whole arm internally rotated, the suprascapular and infrascapular muscles are elongated while the pectoral and subscapularis muscles are contracted. Because of this complicated lesion in the first group of cases, Putti carried out the surgical therapy in two stages. First, a Sever operation was done with severance of the contractured muscles and capsule. The arm was put up in abduction and external rotation for two months. Second, by this approach osteotomy was done through the neck of the humerus, and with elbow flexed at 90 degrees the lower part of the arm was internally rotated to a neutral position. With the arm abducted 45 degrees a second shoulder spica was applied and left on for two months. The treatment in the second group of cases consisted primarily of splinting and physical therapy. Primary nerve sutures were performed in a small series of cases with poor results. The treatment in the third group depended on the site of greatest deformity. The illustrations show excellent operative results in the first group of cases.

[ED. NOTE.—This is a splendid, thorough, long article and is well illustrated. Though previously emphasized by authors, the fact that obstetric paralysis of the shoulder is frequently joint trauma essentially and not pure paralysis is often forgotten. This article merits careful reading, and the therapy suggests trial by others.]

PERIPHERAL VASCULAR DISEASE

Intermittent Venous Occlusion for Treatment of Peripheral Vascular Disease.—Collens and Wilensky [13] report the effect of treatment in peripheral vascular disease by means of intermittent venous occlusion.

13. Collens, W. S., and Wilensky, N. D.: The Treatment of Peripheral Obliterative Arterial Diseases, J. A. M. A. **107**:1960 (Dec. 12) 1936.

They used a pneumatic cuff in the lower third of the femur by means of which from 60 to 80 mm. of mercury was applied in alternating periods of two minutes every two hours. In obliterative disease, e. g., arteriosclerosis, relief is obtained, but treatment must be continued indefinitely. Pain was relieved, ulcers were healed and the walking capacity was increased.

NEOPLASMS

Treatment of Giant Cell Tumors of Bone.—Peirce and Lampe [14] report the results of treatment of 40 patients with giant cell tumor of the bone. Five refused treatment and 6 were treated by preoperative irradiation, surgical intervention and postoperative irradiation, 9 by surgical intervention and postoperative irradiation, 4 by surgical intervention alone and 15 by roentgen irradiation alone. The authors conclude that adequate treatment with roentgen rays or adequate surgical intervention offers as much as the two methods combined. Adequate curettage in anatomically accessible areas has given good functional results. Roentgen treatment offers symptomatic relief and often leads to anatomic repair.

Giant Cell Tumor of the Patella.—Sorge [15] was able to find reports of only 15 cases of giant cell tumor of the patella in the literature. He reports 1 additional case. Treatment consisted in complete removal of the patella and a plastic reconstruction of the quadriceps tendon. The functional and cosmetic results were satisfactory.

Synovial Sarcoma.—Synovial sarcoma is a rare form of malignant tumor of the extremities. Knox [16] reports 3 new cases and includes abstracts of 19 reports in the literature. The tumor arises from a joint, bursa or tendon. It has a characteristic microscopic appearance from which a diagnosis can be made; it is resistant to irradiation. No permanent cure by amputation has yet been recorded.

A series of 117 cases of tumor of the bone in children is presented by Goin and Carroll.[17] In 8.6 per cent the growth was malignant. A classification of bone tumors is offered, in which they are placed in three main groups: (1) those arising from osteogenic elements, (2) those arising from tissues resident in the bone and (3) those of metastatic origin. A review of the characteristics of the various types of tumor of the bone is made, and the results of treatment in the authors' cases are outlined. The writers recommend that a biopsy specimen be secured by aspiration in every case in which a tumor is suspected, and

14. Peirce, C. B., and Lampe, I.: Giant Cell Bone Tumor, J. A. M. A. **107**: 1867 (Dec. 5) 1936.

15. Sorge, E.: Riv. di chir. **2**:399, 1936.

16. Knox, L. C.: Am. J. Cancer **28**:461, 1936.

17. Goin, L. S., and Carroll, R. L.: Radiology **27**:261, 1936.

they offer a plan of approach to the diagnosis of the various types of tumor by roentgen examination.

Hodgkin's Disease with Osseous Involvement.—Dresser and Spencer [18] present a series of 66 cases of Hodgkin's disease with involvement of the bone. The greatest number of these occurred in persons in the third and fourth decades, and the most frequent location was in the spine, pelvis and femur. The incidence of involvement of the bones in Hodgkin's disease as demonstrated roentgenographically is considered to be about 10 per cent, although it is found to be higher at autopsy. Involvement of the bones in Hodgkin's disease is usually considered to occur late, but in 16 of the 66 cases studied the lesions caused the presenting symptoms. The roentgenographic picture usually simulates that of a metastatic malignant process, but it may be confused with the roentgenographic picture of Ewing's tumor, osteogenic sarcoma, bone cyst or osteomyelitis. There is never an entirely characteristic roentgenographic picture; therefore, it must be considered in any unusual lesion. Hodgkin's disease with involvement of the spine often causes collapse of the vertebral body, and when the long bones are involved a pathologic fracture may occur. Involvement of bone may be by direct invasion or by metastasis to the blood stream. Primary Hodgkin's disease of bone has never been observed. The response of the lesion in the bone to irradiation is variable and in general is less satisfactory than that of lesions elsewhere. There is often striking relief from pain, although the prognosis is hopeless. In 36 cases the average duration of life after the first symptoms was three and one-half years; the average duration of life in these cases after the lesion of the bone became evident was eleven months.

Sun Ray Hemangioma of Bone.—Anspach [19] discusses a case of hemangioma of the skull in a child of 11 and then considers the roentgenographic appearance of this type of tumor in different bones. In flat bones a hemangioma usually gives a "sun-ray" or "sunburst" appearance of radiating bone spicules. In proved cases of this tumor in the flat bones there have been strikingly similar patterns. In long bones the sun-ray appearance is not as typical or as constant, and the "soap-bubble" appearance occurs about as frequently. In the spine the predominant features are vertical streaks of parallel densities. The author concludes that although hemangioma is not common it must be suspected in any case of bone tumor showing "sun-ray" spicules, whether occurring in flat or in long bones. No tumor of this sort should be removed without a biopsy first. Treatment should be conservative and consist of roentgen or radium therapy.

18. Dresser, R., and Spencer, J.: Am. J. Roentgenol. **36**:809, 1936.

19. Anspach, W. E.: Sunray Hemangioma of Bone, with Special Reference to Roentgen Signs, J. A. M. A. **108**:617 (Feb. 20) 1937.

MISCELLANEOUS

Ischiopubic Osteochondritis.—Torgersen[20] reports 2 instances of ischiopubic osteochondritis in children 8 and 9 years old. Roentgenograms showed slight swelling and sclerosis of the junction between the lower ramus of the pubis and the ischium. The symptom was pain in the inguinal region on walking. The author believes that mechanical factors played a part in causing the lesion. The treatment consisted in preventing overexertion and in correcting static defects. The literature on this lesion is reviewed.

Cervical Rib and Anterior Scalenus Syndrome.—Flothow[21] reports irritation of the brachial plexus from abnormalities in the supraclavicular triangle. Whether cervical ribs are present or not, section of the anterior scalenus muscle has given relief and is safer than removal of the cervical ribs.

New Roentgenograms of the Skeleton.—Zwerg[22] mentions new positions for obtaining valuable roentgenographic information on bones. These are: (1) a transthoracic lateral view of the upper end of the humerus, (2) a transpelvic lateral view of the upper end of the femur, the other hip being flexed 90 degrees to get it out of the field, (3) a lateral view of the cervical portion of the spine, taken at a distance of 6 feet (183 cm.), a flat casette being used, and (4) a view of the sternum and patella taken with the patient prone rather than supine.

Disk-Shaped Lateral Meniscus with Snapping Knee.—Four cases of disk-shaped lateral meniscus are described by Middleton.[23] He attributes the snapping noise which is associated with movement in this condition to the presence of a ridge dividing the disk into anterior and posterior facets, which contact with the femoral condyle in extension and flexion, respectively. When flexion is being carried out the ridge is pushed back till the movement is almost complete, when it escapes forward with an audible click. The reverse movement of the disk takes place during extension. When the disk-shaped meniscus is fractured, the click takes place over the fractured edge.

Os Acromiale and Its Significance.—The os acromiale has long been recognized by anatomists as an ununited end of the acromium. Schär and Zweifel[24] report 22 cases of this anomaly. Occasionally local pain is felt either spontaneously or after trauma. The condition is usually on the right side and is more frequent in the male. Its medicolegal significance lies in its resemblance to an old fracture line. Roentgeno-

20. Torgersen, J.: Norsk mag. f. lægevidensk. **97**:951, 1936.
21. Flothow, P. G.: West. J. Surg. **44**:570, 1936.
22. Zwerg, H. G.: Arch. f. klin. Chir. **185**:578, 1936.
23. Middleton, D. S.: Brit. J. Surg. **24**:246, 1936.
24. Schär, W., and Zweifel, C.: Beitr. z. klin. Chir. **164**:101, 1936.

grams of the axillary region with the humerus in 90 degrees of abduction reveal the anomaly clearly.

[Ed. Note.—There is still confusion regarding the diagnosis of this lesion, often by expert roentgenologists. The lesion is not rare, and attention should be called to it.]

Anomalous Bones of Wrist and Foot.—Watkins [25] lists and describes 12 anomalous conditions in the wrist and 12 in the foot which might be wrongly considered as due to injuries to these parts. The most important and confusing anomalies in the wrist were congenitally bipartite scaphoids and congenitally ununited styloids of the ulna. In the foot the most important variations were the os trigonum, the tibiale externum (separate and unfused tuberosity of the scaphoid) and a secondary os calcis.

Osteopoikilosis.—Holly [26] reports 4 cases of osteopoikilosis. Roentgenograms showed multiple discrete symptomless small areas of increased density in the bones of the extremities. The lesions occurred in a mother, a daughter and two sons, who were studied for five years. The author concludes from his observations that, as previously shown, the disease is familial. The islands of condensation are not constant; in the adult they may entirely disappear. In the child new areas appear with the growth of bone, which may entirely disappear as the child grows or may increase in size as bony development progresses. There is no variation in the uric acid content of the blood or the calcium-phosphorus metabolism.

ORTHOPEDIC OPERATIONS

Tendon Transplantation in Radial Paralysis.—Bonola [27] describes 3 cases of old paralysis of the radial nerve in which the treatment consisted of transplantation of tendons from the antagonistic muscles. All of the old operative procedures are reviewed. In the 3 cases described the operation consisted of modified insertion of the flexor carpi radialis longus tendon into the extensor tendons of the thumb and fingers. Usually the flexor carpi ulnaris tendon is inserted into the extensor tendons of the second to the fourth finger and the flexor carpi radialis tendon into the abductor longus and extensor tendons of the thumb. One case is illustrated in which apparently good results were obtained.

Hallux Valgus; Comparison of Two Operations.—Lloyd [28] reports 20 cases of bilateral hallux valgus in which different operations were performed on the two sides. On one side the head of the metatarsal

25. Watkins, W. W.: Anomalous Bones of Wrist and Foot in Relation to Injury, J. A. M. A. **108**:270 (Jan. 23) 1936.

26. Holly, L. E.: Am. J. Roentgenol. **36**:512, 1936.

27. Bonola, A.: Chir. d. org. di movimento **22**:239, 1936.

28. Lloyd, E. I.: Brit. J. Surg. **24**:341, 1936.

bone was excised, and on the other, the base of the phalanx and the exostosis of the metatarsal bone. From the patient's replies at the end of from one to three years, it seems there is no choice between the operations as a means of relieving the pain of hallux valgus.

A New Operation for Hallux Valgus.—According to Girdlestone and Spooner,[29] hallux valgus consists of valgus of the proximal end of the phalanx and varus of the first metatarsal bone, with splayfoot. The commoner types of operation for hallux valgus are: (1) resection of the head of the first metatarsal bone and (2) hemiphalangectomy, which reduces the deformity but does not improve the splayfoot or the metatarsalgia, if present. These difficulties may appear postoperatively, if not present before. For this reason a new operation was devised, consisting in the removal of the exostosis, removal of the cartilage from the proximal end of the phalanx and removal of all of the phalanx except the very base containing the attachments of the adductor hallucis muscles. This small block of bone is fastened as far medial as possible on the head of the first metatarsal bone with a beef bone peg. This operation improves or prevents splayfoot as well as relieves the hallux valgus and hallux rigidus. Ten of 20 patients with hallux valgus treated by subtotal phalangectomy complained of metatarsalgia and splayfoot postoperatively and 3 complained of them preoperatively. Thirteen of 30 patients who were operated on by the new technic had metatarsalgia preoperatively, but only 1 complained of it postoperatively.

FRACTURES

Metge [30] describes an avulsion fracture of the spinous process which occurs while the patient is working. Detachment of the spinous process of the seventh cervical vertebra is the most common form; less commonly the first or second dorsal spinous processes are involved. The lesion occurs when a workman bends his head backward while he is hurling a heavy shovelful of earth upward onto a truck. It has long been described as a clinical syndrome which is often incorrectly diagnosed. Roentgenographic study reveals the lesion if the plates are properly exposed with the lesion in mind. The author finds that excision of the avulsed spinous process results in the quickest and surest recovery.

Anomalies and Fractures of the Vertebral Articular Processes.—Bailey [31] describes and differentiates between anomalous fissures across the vertebral articular processes and fractures in this region. Anomalous centers of ossification at the tips of the articular processes are more

29. Girdlestone, R. G., and Spooner, H. J.: J. Bone & Joint Surg. **19**:30, 1937.

30. Metge, E.: Deutsche med. Wchnschr. **62**:1922 (Nov. 20) 1936.

31. Bailey, W.: Anomalies and Fractures of Vertebral Articular Processes, J. A. M. A. **108**:266 (Jan. 23) 1937.

frequent than commonly supposed. In half the 10 cases described the history of trauma was vague or entirely absent. The defect was often bilateral, and rarely were the superior processes affected. Fracture of the articular process without other injuries to the spine was rare. Pain was prompt and severe, and the fragments were usually displaced.

Ununited Fractures of the Shaft of the Humerus.—The end-results in any large series of ununited fractures treated by operative methods are rarely reported. Campbell [32] reports the end-results in 50 cases of ununited fracture of the shaft of the humerus. One patient with an oblique fracture line was treated by transfixion of the fragments with autogenous bone pegs, solid union resulting. The remaining 49 were treated by the application of a massive onlay graft from the tibia secured by autogenous bone pegs. In addition to this, a strip of endosteum was placed within the medulla and bony shavings were placed about the site of the fracture. There were 3 failures in the 49 patients so treated. There were no failures in the 4 patients in whom postoperative infection occurred.

[ED. NOTE.—The results in this series of cases are excellent. The high percentage of successful unions would indicate that this is the method of choice in treating nonunion of the shaft of the humerus and that less secure means of fixation should be discarded.]

Results of Fractures of the Olecranon.—Laukka [33] treated 103 fractures of the olecranon and was able to study the end-results in 56 cases. Five fractures were compound. Loose fragments in the elbow joint were seen in 17 patients. Thirty-five patients were treated by immobilization alone. This was used chiefly in children in whom there was separation of not more than 5 mm. and nothing prevented complete apposition of the fragments. Sixty-five patients were operated on. In most cases stainless steel wire was used to hold the fragments together, and in a few cases, silk. In the follow-up study 10 of 11 patients were found to have less than 20 degrees limitation of motion. Good function was seen in all of these patients. Less than 20 degrees limitation of motion was seen in 22 of 33 patients. Pseudarthrosis was found in 6 patients. Arthritis deformans was frequently seen one or two years after the fracture in 36 per cent of those operated on and in 13 per cent of those treated conservatively. Fracture of the head of the radius was a complication in 7 patients.

Fractures of the Neck of the Femur.—Magnuson [34] attempted to evaluate the various methods of treatment used for fracture of the

32. Campbell, W. C.: Ann. Surg. **105**:135, 1937.

33. Laukka, E.: Acta chir. Scandinav. **79**:93, 1936.

34. Magnuson, P. B.: Fracture of Neck of Femur: Evaluation of Various Methods Advanced for Treatment, J. A. M. A. **107**:1439 (Oct. 31) 1936.

neck of the femur. A fracture in this region should be classified according to its line and plane and according to the amount of displacement which originally occurred. When there is considerable obliquity of the fracture line, visual reduction is preferable with immediate fixation. Each patient presents an individual problem. While the Whitman, the Leadbetter and the well leg traction method have their place, internal fixation of the fragments gives greater comfort and makes nursing care easier and the chances of union greater. The author feels that close bony contact, anatomic apposition and absolute fixation are the three most important factors in securing union.

"March Foot."—Elward [35] reviews the literature on "March foot" due to foot strain and fracture of a metatarsal bone. He describes two varieties, one due to sudden strain of the foot with fracture of a metatarsal bone and the other due to chronic foot strain and fracture of a metatarsal bone with no definite history of trauma. In the roentgenograms one must differentiate chiefly between sarcoma and Köhler's disease. In the absence of trauma, sarcoma may be suspected, but roentgenograms do not show the "sun-ray" structure. In cases in which the diagnosis is doubtful, weekly roentgenographic examination should make the diagnosis clear. Conservative treatment is indicated.

Fractures of the Neck of the Femur in Childhood.—Mitchell [36] reports 10 cases of fracture of the neck of the femur in children. He divides the lesion into three types: (1) epiphysial separation, (2) transcervical fracture and (3) cervicotrochanteric fracture. Bony union practically always occurs, often with coxa vara. The author advises treatment by continuous traction with the hip immobilized in abduction.

Traumatic Separation of the Upper Femoral Epiphysis.—Pfeiffer [37] adds 3 cases to the list of cases of traumatic separation of the upper femoral epiphyses during childbirth. The lesion is not easy to recognize. Crepitus is usually absent. Swelling or limitation of motion of the hip generally calls attention to the injury. Careful roentgenographic study will reveal displacement of the intact shaft early and bony callus later. Early recognition, replacement and adequately long fixation are essential if traumatic coxa vara is to be avoided.

Fractures in the New-Born.—A review of the last 6,000 cases of fracture at the Boston Children's Hospital reveals 115 birth fractures, 34 per cent of which were not recognized at the time of their occurrence. In this series the fractures were distributed as follows: fracture of the

35. Elward, J. F.: Am. J. Roentgenol. **36**:188, 1936.

36. Mitchell, J. I.: Fracture of Neck of Femur in Children, J. A. M. A. **107**: 1603 (Nov. 14) 1936.

37. Pfeiffer, R.: Beitr. z. klin. Chir. **164**:18, 1936.

clavicle, 53; of the humerus, 38; of the femur, 20, and of the tibia, 4, including 3 of the fibula also. Unusual obstetric maneuvers were required for 20 per cent of the fractured clavicles, 92 per cent of the fractured humeri, 72 per cent of the fractured femurs and 50 per cent of the fractured tibias. Thorndike and Pierce [38] believe that adequate treatment of fractures in the new-born should consist in: (1) early diagnosis, (2) prompt realinement of the fragments (healing is so rapid that after five days it is impossible to reduce the fractures manually), and (3) proper fixation until union has taken place. Methods of treatment are explained in this article: A skein of yarn with figure eight traction is recommended for a fractured clavicle. It was found that in 100 per cent of the cases in which fracture of the femur was treated by strapping the femur to the abdomen the result was unsatisfactory; for this reason an overhead frame with adhesive traction is recommended. Not one instance of true intra-uterine fracture was found in this review. In most cases in this series the fracture occurred in a large baby.

Injection of Alcohol into the Intercostal Nerve in Treatment of Fractures of the Ribs.—Rabboni [39] reports 10 cases of fracture of the ribs in which the therapeutic method of Latteri was used. Rabboni states that external splinting does not suffice to immobilize a fractured rib sufficiently to relieve pain and allow normal respiratory movements. If a small amount of alcohol is injected in the vicinity of the corresponding intercostal nerves, nerve block ensues. This is accompanied by complete relief of pain, normal respiration and, without splinting, normal healing of the fractures. If injection into more than two nerves is necessary, the nerves are taken in couples on consecutive days. The procedure of injection consists of inserting the needle at right angles to the undersurface of the rib, and with the needle in contact with the rib it is gently manipulated so that the point is upward just mesial to the inferior ridge of the rib.

[ED. NOTE.—This method of treatment should be used with caution. Unfortunate complications have followed failure to splint broken ribs. Injection into the intercostal nerve is also not without danger.]

Procaine Hydrochloride in the Treatment of Articular Fractures.—In cases of fracture in which there is little or no displacement and in which maintenance of position with apparatus is unnecessary, Leriche and Froehlich [40] have been starting active motion of the adjacent joint immediately. They first inject 1 per cent procaine hydrochloride into

38. Thorndike, A., and Pierce, F. R.: New England J. Med. **215**:1013 (Nov. 26) 1936.

39. Rabboni, F.: Chir. d. org. di movimento **22**:263, 1936.

40. Leriche, R., and Froehlich, F.: Presse méd. **44**:1665 (Oct. 24) 1936.

the soft tissues about the fracture and if possible into the fracture fissure itself. The effect of this injection lasts for from twelve to twenty-four hours, and the treatment is repeated daily for from two to three weeks. Active motions are repeatedly performed while the parts are under the anesthesia. The authors believe that restoration of function is greatly hastened by this method. In 2 of the reported cases there was gross displacement of the radial head. No attempt was made to reduce or remove the head, but excellent function was obtained in each case by the method described.

[ED. NOTE.—Fracture without displacement and needing no fixation usually gives little trouble however treated. In the foregoing cases of fracture of the head of the radius, both marked displacement and restoration of function without removal of the head seem extraordinary. It does not seem likely that such results could be obtained uniformly.]

RESEARCH

Studies in Healing of Fractures.—Krull [41] studied the problem of healing of fractures with cultures of tissue from chick embryos. At ten days the anlage of the femur was freed from the surrounding tissue. The femur was twisted, and the broken pieces were placed in dishes or flasks containing a mixture of chicken plasma and embryonal extract. Different mineral concentrations and temperatures were investigated. While fibroblastic repair was seen, no calcification or bone production occurred.

Thymus and Bone Regeneration.—Portions of the radius were resected by Haas and Hanke [42] in rabbits, and healing was observed in a group of animals from which the thymus had been removed. This group did not show any essential difference in bone formation from the control group. The feeding of thymus extract to the animals after the removal of the thymus had no effect either. The rabbits chosen were all growing young animals.

Thyroid Activity and Healing of Fractures.—Eitel and Lexer [43] administered thyroid preparations to rabbits. After fracture of the humerus, administration of thyroid was continued, and at various dates the rabbits were anesthetized and mercury was injected into the axillary artery of the fractured extremity. Study of the visualized blood supply of the sites of the fractures indicated that the administration of thyroid caused hyperemia in the area of the callus to occur earlier and that the callus was stronger and lasted longer than in the control animals.

41. Krull, G.: Arch. f. orthop. u. Unfall-Chir. **37**:131, 1936.
42. Haas, A., and Hanke, H.: Deutsche Ztschr. f. Chir. **247**:724, 1936.
43. Eitel, H., and Lexer, E. W.: Arch. f. klin. Chir. **185**:587, 1936.

Influence of the Mineral Constituents of Bone on Ossification.—As a result of carefully controlled animal experimentation, Bisgard [44] concludes that the synthetic bone salts calcium carbonate, calcium phosphate and magnesium phosphate when locally deposited had no influence on osteogenesis or ossification, whereas bone in the form of partially viable bone, boiled bone or bone ash had a favorable influence on ossification when deposited locally.

Metastases from Tumor Transplants.—Schopper [45] reports the results of studies on osseous metastases from tumor material injected intravenously into rabbits. The Brown-Pearce [46] rabbit tumor was used. The rabbits were killed at varying intervals. The metastatic tumors showed best in from three to four weeks. Proliferating tumors were found often as foci without any marked reaction in the surrounding tissue; sometimes there was destruction of bone; rarely was there new bone formation. Tumors were found in the bone marrow and under the periosteum, chiefly in the region of the metaphyses of the long bones. The periosteal tumors were primary transplants and not extensions from the marrow cavity. They stimulated osteophytic activity and at the same time destroyed bone by pressure. New bone formation occurred chiefly by laying down layers of new bone. Rarely was there a metaplasia of the surrounding stroma. No deformities or pathologic fractures were observed.

Study of the Supraspinatus Tendon in Cadavers.—Skinner,[47] on the basis of dissections on cadavers, presents a description of changes occurring in the supraspinatus tendon in response to continued trauma and to age. It would appear that about 20 per cent of all adult shoulders show some degree of change in the supraspinatus tendon, of which one fourth will show some degree of rupture or splitting. Because of its anatomic relations, the supraspinatus tendon is subject to changes consisting of alteration from fleshy fibers ending in a short tendon to a wide aponeurosis of fibrous tissue blending with the infraspinatous tendon. When this has occurred, because of weakness, splitting, rupture or calcification may follow. Associated changes occur in the greater tuberosity, the intertubercular sulcus, the articular cartilage and the tendon of the long head of the biceps muscle.

Vascularization and Pathologic Structure of the Cotyloid Ligament.—An excellent study of the circulation of the acetabulum is given by Logroscino and Dotti.[48] The blood supply is derived from the superior

44. Bisgard, J. D.: Ossification: Influence of Mineral Constituents of Bone, Arch. Surg. **33**:926 (Dec.) 1936.

45. Schopper, W.: Virchows Arch. f. path. Anat. **298**::527, 1937.

46. Domagk, G.: Centralbl. f. allg. Path. u. path. Anat. **61**:108, 1934.

47. Skinner, H. A.: J. Bone & Joint Surg. **19**:137, 1937.

48. Logroscino, D., and Dotti, E.: Chir. d. org. di movimento **22**:285, 1936

and the inferior gluteal vessels and from the obturator. Because of the blood supply, localization or infectious lesions and trophic changes following trauma may be explained.

[ED. NOTE.—This article is too long and full of data to abstract, but it merits reading by all interested in the anatomic and pathologic structure of the acetabulum.]

Ligamentum Teres and Its Vessels.—Schwaiger [49] studied the ligamentum teres femoris grossly and microscopically at various age periods. He found that vessels could be found in the ligamentum teres up to old age. From the age of 2 years upward there was progressive obliteration of the blood vessels. He believes that the importance of the vessels in the ligamentum teres has been overestimated. No pathologic changes were observed in the cartilage of the upper end of the femur or the surrounding tissues from obliteration of the vessels in the ligamentum teres.

Correlation of Pathologic and Roentgenographic Findings in Tuberculous and Pyogenic Infection of the Vertebrae.—Various authors have assumed that tuberculous or pyogenic hematogenous infection may localize first in the intervertebral disk. Compere and Garrison [50] point out the fact that the adult intervertebral disk has no blood supply and therefore the disk is never the site of primary infection but is affected by secondary invasion from some adjacent vertebral focus. In a pathologic study of 4 cases of uncomplicated Pott's disease they found that the primary focus was in the vertebral body, either in the centrum or near the epiphysial ring. The disk tissue acted in similar fashion to articular cartilage in other joints and was resistant to destruction. The disease spread by extension beneath the longitudinal ligament and not directly through the disks. The narrowing of the intervertebral space commonly noted seemed to be due to extrusion of the nucleus pulposus through a weakened annulus fibrosus or cartilage plate. In two cases of pyogenic osteomyelitis pathologic examination showed rapid destruction of the adjacent intervertebral disk, as joint cartilage is rapidly destroyed by the proteolytic enzymes in the pyogenic exudate. In contrast to tuberculous infection, regeneration of bone and ankylosis of the vertebral bodies are much more likely to occur in pyogenic infection. The results of microscopic examination in 3 cases of mixed infection (tuberculous plus pyogenic) of the spine were reported.

Rôle of the Reticulo-Endothelial System in the Deposition of Colloidal Matter in Articular Cavities.—In studying the rôle of the reticulo-endothelial system in the deposition of colloids and particulate

49. Schwaiger, M.: Ztschr. f. Orthop. **65**:297, 1936.
50. Compere, E. L., and Garrison, M.: Ann. Surg. **104**:1038, 1936.

matter in articular cavities, Kuhns and Weatherford [51] used rats into which they injected trypan blue subcutaneously or by gavage. Various series of experiments were made in an attempt to exclude the rôle of the lymphatics. In conclusion, the authors found that colloidal and particulate matter was carried to the articular cavities from the various tissues of the body, such as the skin and the gastro-intestinal tract, through the blood stream. This material was stored chiefly in histiocytes in the synovial membrane, in the bone marrow and in lesser amounts in the intermuscular septums and in the articular fat pads. Mild inflammation in articular tissues tended to increase the amount of deposition of such transplanted substances. Local blocking of the reticulo-endothelial system was transitory and incomplete and was not effective in preventing the deposition of colloidal and particulate matter in articular tissue.

Auscultation of Joints.—Steindler [52] stresses the importance of auscultation in the examination of joints. He presents a preliminary report based on the auscultatory findings in a series of 397 knee joints. The instruments used were the stethoscope, the cardiaphone and the oscillograph. The interpretation of sound phenomena is difficult because extraneous and extra-articular noises must be differentiated from the intra-articular noises. However, certain sounds can be considered intra-articular because of their character and the regularity of their occurrence. Sustained grating in all quadrants of the knee joint indicates general involvement of the joint with arthritic changes which may or may not be the background of isolated internal derangements. Isolated sounds are cracks, clicks and thuds. High-pitched cracks indicate a hard body, such as a joint mouse, while lower-pitched clicks and thuds indicate softer bodies, such as fringes and semilunar cartilages. The author does not claim to be able to make definite diagnosis by auscultation but feels that it is of aid in locating the lesion inside the joint.

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51. Kuhns, J. G., and Weatherford, H.: Rôle of Reticulo-Endothelial System in Deposition of Colloidal and Particulate Matter in Articular Cavities, Arch. Surg. **33**:69 (July) 1936.

52. Steindler, A.: J. Bone & Joint Surg. **19**:121, 1937.